New Orleans Medical
and
Surgical Journal

Vol. 101 AUGUST, 1948 No. 2

POSTRABIES VACCINE PARALYSIS*

SHEA HALLE, M. D.†
NEW ORLEANS

In 1885 Galatier and Pasteur demonstrated the infectious origin of rabies. Pasteur shortly thereafter developed a prophylactic treatment against the disease. Since then this preventive has been employed almost universally, but with its introduction has come a complication—the possibility of postrabies vaccine paralysis. Recently the seriousness of these complications was forcibly presented to us when two patients were admitted to the Veterans Administration hospital with encephalomyelitic lesions following vaccine treatment. In each instance the indication for the Pasteur treatment was questionable. The history relating to both of these patients reveals the presence of large numbers of rabid animals in the vicinity, and the promiscuous use of the vaccine for other individuals in the community. Because of these circumstances, the following two cases are being reported, and it is our belief that a discussion of the types, incidence, etiology and therapy of postvaccinal reactions, and indications for application of the Pasteur treatment, is invited.

CASE REPORT

Case No. 1.

A twenty-four year old white male was admitted to the hospital complaining of weakness of all extremities, and inability to void.

Fifteen days prior to admission the patient was bitten by his dog. Within twenty-four hours the dog died and the patient was started on antirabic vaccine. Study of the dog's brain was not carried out. A local physician used a reputable Semple-type vaccine for immunization. A detailed review of the injections received reveals: First day: Immediate reaction, around the site of injection in the abdominal wall, consisted of a red tender "lump" in skin. This subsided in a few hours. Second day: Same. Third day: After this injection the patient developed chills, fever and headache; these subsided overnight, and the injections were continued. Fourth to tenth days: During this week the patient had severe malaise, headache and drowsiness, with an exacerbation following each injection. After the tenth injection the headache became severe; the neck was painful especially on movement. Backache increased in intensity, and chills and fever occurred several times. The eleventh injection was given in bed because the patient was unable to get up. Shortly thereafter he could not move his limbs, was unable to void or defecate voluntarily, and became very drowsy. He was catheterized following the eleventh injection. On succeeding days two more injections were given, but the patient has no recollection of these, or other incidents following the tenth injection. A total of thirteen injections was administered.

Two days after completion of the series, the patient was brought to this hospital for treatment.

Physical Examination: The patient was a semi-stuporous white male, well developed and well nourished, apparently seriously ill, and poorly responsive to questioning. Blood pressure 130/80. Pulse 95. Temperature 100.0° F. Respiration 25. There was marked herpes labialis, and the abdomen was diffusely tender to deep palpation. Other positive physical findings were neurological. Cranial nerve functions were intact except for transient diplopia and slurring of speech. The motor system revealed generalized weakness of all extremities, with moderate flaccidity. Some

*Published with permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the author.

†Resident in Medicine, Veterans Administration Hospital, New Orleans, Louisiana.

function remained in thighs and toes (slightly more in upper extremities), but there was marked loss of ability to coordinate motions of the hands and upper extremities. Deep reflexes originally were diminished in left upper extremity, both ankles, and left knee. Abdominal reflexes were bilaterally absent. There was suggestive Babinski bilaterally. Tests of sensory perception revealed spotty areas of hypesthesia above TIO, and spotty areas of hyperesthesia below this level. Also observed were inability to void or defecate. The anal sphincter was relaxed.

Laboratory Examinations upon admission: Blood: WBC 6400/cu. mm.; RBC 4,250,000/cu. mm. Differential: Neutrophiles 77%; Lymphocytes 21%; Monocytes 1%; Eosinophils 1%.

Urine: Albumin, heavy trace. Sediment, 20 RBC's per hpf.

Lumbar puncture: Normal dynamics, clear fluid. Initial pressure 180 mm. water. Cells, 40-30 Lymphocytes, 10 Neutrophiles. Total proteins, 35 mgs.%. Chlorides 742 mgs.%. Sugar 80 mgs.%. Colloidal Gold 0000000000. Wassermann negative. Cultures negative.

Seven days later—Lumbar puncture: Colloidal Gold 2222332200. Cells 55 Lymphocytes/cu. mm. Total proteins 190 mgs.%.

Course in Hospital: The neurologic status changed constantly, with paresis becoming more severe, until all muscle function in the lower extremities was lost. Some function remained in the neck muscles, and in the biceps and deltoids, but little in the forearm or hand. Occasionally, speech became indistinct. Intercostal muscles were undisturbed. Reflexes changed daily, and a hyperactive left knee jerk appeared for one day. Sensation also changed daily, with the hyperesthesia in lower extremities becoming more marked. A retention catheter was placed in the bladder. Fluids were supplemented by parenteral administration. Patient took some nourishment by mouth, despite frequent nausea and vomiting. The acute process gradually subsided over two to three weeks, with the patient becoming more responsive. Diplopia disappeared after two weeks with no further speech changes being noted. Three weeks after admission, patient began to have some return of function in the upper extremities. Physical therapy was then started. A return in the psoas and quadriceps muscle function followed, but legs and feet were totally paralyzed. Fecal impaction threatened on several occasions, and repeated enemas were essential for its avoidance.

There was no indication of returning bladder function, and a severe infection of the lower genito-urinary tract developed because of a retention catheter. Suprapubic cystotomy, therefore, was resorted to about five weeks after admission. Cystograms at this time revealed no marked change in bladder size or shape. The bladder held more than 30 ounces of fluid on several occasions without overflowing. This caused mild discomfort, suggesting hypotonicity of the bladder wall. A month after admission the patient had his first voluntary bowel movement. During the following two months there was gradual return of function and the patient was able to sit up. At the beginning of the month he began to move his toes, and several weeks later motion returned to his ankles and legs. After fourteen weeks he began to void voluntarily, and the suprapubic catheterization was terminated. Urine then revealed gross pus and blood with 2 to 3 plus albumin and a few casts from time to time; cultures revealed urea-splitting organisms. However, once voiding became voluntary, therapy with sulfonamides and streptomycin cleared the genito-urinary infection.

Eight months have passed since this illness. The patient has regained full use of the upper extremities and the left lower extremity. However, the left quadriceps function is very weak and physical therapy is still being administered. Objectively, it appears that the patient will regain almost completely previous muscular power.

Case No. 2.

A twenty year old white male was admitted to this hospital complaining of headache, and inability to walk and void. About two months prior to this admission the patient's schoolmate was bitten by a dog, and one month later became acutely ill and died within three days. It was believed that he died of rabies. The patient, and many of his neighbors (variously estimated as 20 to 60 people), requested the Pasteur treatment from their local physician despite the fact that they had had no contact with the rabid dog, and only casual contact with the schoolmate. Antirabic injections were begun on the 21st of August and continued with daily injections until the 31st of August. After the seventh day, he complained of malaise, headache, and slight fever, as well as edema, induration and soreness at the site of injection. Injections were, nevertheless, continued until the time the patient was unable to walk; he then decided to enter a hospital for treatment.

Physical Examination: Pulse 88. Temperature 102.0° F. Patient was emaciated, obviously ill and apathetic. He responded slowly to questioning. There was transient diplopia, nystagmus in all directions, moderate photophobia, and mild stiffness of the neck. All reflexes were bilaterally equal, and there was no disturbance of sensation; however, the bladder was easily palpable at the umbilicus. There was generalized weakness.

Laboratory Examinations: Spinal tap: Clear fluid. Pressure 230 mm. of water. Normal dynamics. Cell count 27/cu. mm. Lymphocytes 97% Protein 56 mgm.%. Complement fixation and gold curve—Negative.

Hemogram revealed 10,250 WBC's and 4.1 million

RBC's/cu. mm. Differential; Neutrophils 87%.
Urine: Positive for acetone.
Sedimentation rate: 24.

Course in Hospital: A few days following admission the tendon reflexes on the left diminished, diminution being most marked in the left lower extremity. Pain and touch sensations were normal. Later this was followed by diminution of reflexes of the right lower extremity, these finally disappearing completely. The cremasteric reflexes disappeared as did the abdominals. There were very slight muscle residuals left in the upper extremities. The lower extremities were completely paralyzed, with all reflexes being absent. A suprapubic cystotomy was done because of inability to void.

Within two weeks after admission the patient began to show improvement. The nystagmus and diplopia disappeared. At that time there was only slight residual movement left in upper extremities. At the present time, which is six weeks after admission the patient has continued to progress, slowly but favorably. Some function has returned to the left upper extremity. It is too early to predict the degree of muscle function that may be expected.

TYPES OF REACTION

There are two general types of reactions to the Pasteur treatment: The first includes skin reactions, localized or generalized. These are common, have no associated mortality, and are no contraindication to further therapy. The second type of reaction is the neurologic, and this group is more serious. Horack[4] has divided the two types into six groups:

Group 1. The prompt development of a generalized urticarial rash which responds to the use of adrenalin, and which usually develops in those who have been sensitized by previous injection of nerve tissue. These cases are relatively infrequent and are without mortality.

Group 2. Delayed reactions of the tuberculin type occurring at the site of injection and characterized by local redness, induration, tenderness, and itching. These are the most common type of local reaction encountered. They occur early during the treatment, but are not of serious import.

Group 3. Reaction of the type described as Group 2, but more severe, and frequently associated with constitutional reactions consisting of headache, low-grade fever, lymph-node enlargement, nausea, and general feeling of malaise. Subsequent injections are apt to cause a flare-up at the previous injection sites. It is these patients that one should observe closely for signs of the development of paralysis, and judge their treatment accordingly. These cases are less common than those described in Group 2.

Group 4. The simple peripheral neuritic form, following a short febrile attack and commonly involving the facial nerve.

Group 5. A dorsulumbar myelitis, usually occurring during the second or third week of treatment, and characterized by the gradual onset of fever, weakness, numbness, tingling of the lower extremities, sphincter disturbances, and terminating in paralysis of the extremities. The local reactions are of the more severe type described under Group 3. The mortality rate is low.

Group 6. Paralysis of the Landry type, which is sudden in onset and is associated with high fever, nausea, insomnia, vomiting, headache, girdle pains, retention of urine, and an ascending paralysis. About one-third of these patients die of bulbar paralysis. Local reactions are those of Group 3.

INCIDENCE

The incidence of paralytic reactions varies markedly from place to place, even when similarly prepared vaccine is used. Large series, such as that compiled by McKendrick[6] (League of Nations Rabies Conference) show an incidence of from 1 in 3398 ("live" vaccine) to 1 in 8887 ("killed" vaccine), with almost two million patients treated. Smaller American series show a ratio of 1 in 450 (Hygienic Clinic of Public Health),[5] 1 in 5000 (Georgia),[11] and 1 in 1200 (North Carolina).[4] American groups generally use "killed" vaccine, ordinarily prepared by a modification of the Semple technic. Differences in preparations used are believed unimportant so far as results are concerned. However, they do affect the number of reactions. The great discrepancy in the number of reactions reported above from various treatment centers may be the result of poor reporting of complications in the larger series. However, Charity Hospital of Louisiana in New Or-

leans has treated 7,000 patients with rabies vaccine, with no known paralytic complication. Charity Hospital vaccine is prepared by a modification of the Semple technic.[7, 8]

If one compares the incidence of postrabies vaccine paralysis cited above with the incidence of rabies following treatment, one finds in McKendrick's large series the statistics as enumerated in Table 1.

TABLE 1

MORTALITY FROM RABIES FOLLOWING ANTIRABIES TREATMENT OF MAN

No.	(McKendrick, 1940) Exposure		Per cent mortality	Proportion : 1 in
34,465	Contact; no bite	Animal proved or suspected rabid	0.0005	17,235
26,766	Head bite		1.29	77.5
228,370	Arm bite		0.15	676
18,003	Trunk bite		0.02	4,500
167,400	Leg bite		0.05	2,100

These statistics reveal the grave significance of head bites and arm bites, and the lesser significance of contact with no bite, and leg and trunk bites. Charity Hospital has had eight deaths from rabies in 7,000 cases, an incidence of slightly more than 1 in 1000.[7]

ETIOLOGY

There has been little agreement as to the cause of the reactions described. At first, it was suspected that they were due to an atypical or modified type of paralytic rabies; then a case was found in which paralysis followed treatment although the dog involved was not rabid. In later years, other possible causes implicating the vaccine have been entertained. The street virus used in preparing the vaccine was one of these; another was that toxic material from the rabbit cord, or a neurotropic substance in the rabbit cord was at fault. Bassoe and Grinker[1, 3] presented the possibility of an unknown virus latent in the patient's central nervous system, aggravated by the vaccine. This, they feel, would explain the entire group of postvaccinal and nonvaccinal disseminated encephalomyelitic reactions which pathologically are alike. Smallpox vaccination and the exanthematous diseases such as measles, chickenpox, mumps, smallpox have all been associated with encephalomyelitic syndromes similar to those discussed in this paper.[3]

An impressive possibility at this time is an allergic basis for the reaction. Horack[4] has reviewed this possibility and in a special study found marked allergic histories in patients who had shown reactions. In most patients the disease started during the second or third week of treatment and was preceded by varying grades of local reaction, or general reaction similar to those in patients showing sensitivity to foreign protein injection. Under such circumstances edema, erythema, induration, and pain are often found at the local site of injection. Also there may be urticaria and other constitutional symptoms of general body reaction. Support of this contention is brought out in Seller's series, in which it was shown that five of the seven paralytic reactions occurred in patients who had taken a previous course of the Pasteur treatment.[12] In addition, Schwentker and Rivers[10] found experimentally that brain tissue can act as an antigen and produce organ specific antibodies—at least in rabbits. They conclude that paralytic reaction following antirabies vaccination probably is associated with the development of specific antibodies for brain.

INDICATIONS FOR THE PASTEUR TREATMENT

Although many capable observers question the value of the Pasteur treatment,[14] there are few who oppose its use, at this time, in any patient exposed to rabies, a disease which has been shown to be a uniformly fatal one. However, those with most experience in such therapy are conservative in prescribing specific treatment. Sellers, after long, close observation, believes that the Pasteur treatment should be used only "for persons known or suspected to have been bitten by rabid or suspicious animals."[12] No cases of rabies developed in his series in persons known not to have been bitten. This is true also of large Alabama[2] and Louisiana[5] series in which injury by the teeth of rabid animals accounted for all the deaths. In Table 1 among those with history of exposure other than bite, only one person in 7235 developed rabies. Therefore "exposure" by getting saliva on the bare skin or even in contact

with pre-existent abrasions or by handling the animal[11, 12] is not an indication for treatment. The risk of serious treatment reaction is greater in non-bite cases than the risk of developing rabies. Children, however, are usually treated for contact because of the poor history obtainable, and also because of the lesser danger of reaction from the vaccine. Few postvaccinal paralytic reactions have been reported in children under 15 years.[8]

A major problem involves the bite by a stray dog which cannot be found. Here treatment usually should be given, especially in areas where there is a large reservoir of rabies in the dog population. The public must be taught to catch dogs that have bitten, and never to kill such dogs until a two weeks' observation period is over. In one of our cases the dog died soon after biting the patient. No attempt was made to examine the brain for rabies. It is possible that the dog died from some other cause.

In persons bitten by a dog that can be observed, there is no danger in waiting up to two weeks before giving the vaccine therapy for all bites except those involving the head and upper arm. Because of the high incidence of rabies following bites on the head and upper arm, it is generally accepted to start treatment immediately. The treatment can be interrupted if observation finds the dog involved to be free of rabies.

Most trouble comes from the great fear of rabies in the community. A panicky community in Louisiana recently demanded the Pasteur treatment for many of its citizens after a young man had died of rabies there. Our case, listed second in the foregoing, is the result of this mass demand for treatment. The demand was made despite the fact that nobody had been bitten by the dog except the one who had died of rabies. The public should be warned that the Pasteur treatment is not without danger; that the physician must make the decision as to its use.

DESENSITIZATION

Once treatment has been decided upon, the question of desensitization technic to avoid reaction arises. Horack[4] urges a complete allergic history and the testing with a small dose of diluted vaccine before starting treatment; then, if the patient is sensitive he urges desensitization by the use of graduated doses until the full dose can be given. If a reaction occurs during the course of treatment he again resorts to desensitization before going on. Further details are available in his article.[4] However, most treatment centers, like Charity Hospital, have not resorted to desensitization technics. Pizzolato,[9] working at the Charity Hospital Pasteur Clinic for three years, skin-tested all patients with a history of either *allergy* or a *previous course of treatment*. He found no evidence of a positive skin reaction to the vaccine in either group. This suggests that skin-testing is not helpful. However, skin-testing should be done before giving repeated courses of antirabies treatment. Up to the present time there have been no paralytic reactions in Charity Hospital's Pasteur Clinic.[7]

There are two situations in which desensitization is probably worthwhile. One is the case of a dog bite by a rabid animal, especially on the face or hands, with the development of a paralytic reaction during therapy. In these cases great danger of rabies is still present; if treatment is to continue it is probably wise to desensitize first. The other situation involves the patient who has had the Pasteur treatment previously. The Georgia series[12] warns of the grave dangers of reactions in these cases. As already mentioned five of seven paralytic reactions occurred in patients receiving their second course of treatment. Sellers used a five-to-six day treatment course given very cautiously in these repeaters. He considers this a booster to immunity mechanisms produced by a previous course of treatment. He also recommends stopping this booster course at the slightest hint of trouble—even marked *local* reaction.

Every patient applying for antirabic vaccine therapy must be individualized. The physician, not the patient, must decide if the therapy is needed since the patient

cannot appreciate the danger associated with the treatment.

TREATMENT OF REACTION

Little can be added to the known treatment of these encephalomyelitic complications. Profiting from experience in our first case, a suprapubic cystotomy was done early in the second case. This procedure should probably be used if paralysis of bladder and muscles suggests long delay in return of function.

Both of our cases were started on antihistaminic drugs fairly early, but no significant results were obtained. A nourishing diet, parenteral fluids, vitamins, physical therapy and general supportive care were given; fecal impactions were avoided only by vigilant care.

DISCUSSION

Although in this paper we have concerned ourselves primarily with paralytic reactions to the Pasteur treatment, the real problem is the eradication of rabies. This can only be accomplished by an active campaign against reservoirs, chiefly dogs and cats, in the community. By *destroying strays* and muzzling or restraining pets, this menace can be controlled. Other possible measures are immunization of dogs against rabies, and strict dog-quarantine laws. Especially is this important now in view of the rising incidence in the past few years of rabies in animal brains examined at various centers in Louisiana.[7, 8]

For the present though, when giving the Pasteur treatment, the indication should be considered carefully. The patient should be individualized. If patients continue to insist on treatment, it may be wise to restrict the use of the vaccine to local public health or hospital centers. Among the advantages would be more accurate reporting of treatment results, failures, and reactions. Also, the number of exposures other than real bites that are treated would be decreased.

Finally, the use of desensitization technic should be considered in the presence of marked allergic history for severe reaction from injections. The grave danger from more than one course of treatment should be kept in mind.

SUMMARY

1. Two case histories of encephalomyelitic reactions following the Pasteur treatment are presented.

2. The etiology and incidence of these reactions are reviewed.

3. Indications for the use of the Pasteur treatment are considered.

4. Desensitization technics and the treatment for paralytic reactions are mentioned.

REFERENCES

1. Bassoe, P. and Grinker, R. R.: Rabies and rabies vaccine encephalomyelitis, Arch. Neurol. & Psychiat., 23:1280, 1930.

2. Denison, G. A. and Dowling, J. D.: Rabies in Birmingham, Alabama. Human mortality as affected by antirabies treatments, J.A.M.A., 113:390, 1939.

3. Grinker, Roy R.: Neurology, Charles C. Thomas, Illinois, 1944.

4. Horack, Harold M.: Allergy as a factor in the development of reactions to antirabic treatment, Am. J. Med. Sci., 197:672, 1939.

5. McCoy, G. W.: Antirabic vaccine paralysis, Public Health Reports, 45:1888, 1930.

6. McKendrick, A. C.: A ninth analytical review of reports from Pasteur Institutes on the results of antirabies treatment, Bull. Health Org. League of Nations, 6: No. 1, 31, 1940.

7. Moss, E.: Personal Communication.

8. Palik, E. E. and Moss, E. S.: Rabies. A ten-year survey of the Pasteur Institute of Charity Hospital of La. at N. O., New Orleans M. & S. J., 97:153, 1944.

9. Pizzolato, P.: Personal Communication.

10. Schwentker, F. F. and Rivers, T. M.: The antibody response of rabbits to injections of emulsions and extracts of homologous brain, J. Exp. Med., 60:559, 1934.

11. Sellers, T. F.: Antirabic treatment, J. M. A. Georgia, 28:298, 1939.

12. Sellers, T. F.: Limitations of antirabic treatment, J. M. A. Georgia, 35:132, 1946.

13. Thomas, C. R.: Complications following the use of antirabies vaccine with suggestions as to treatment, South. M. J., 37:539, 1944.

14. Webster, L. T.: Rabies, The MacMillan Company, New York, 1942.

THE NECESSARY PERSONAL ORAL HYGIENE FOR PREVENTION OF CARIES AND PERIODONTOCLASIA*†

CHARLES C. BASS, M. D.

NEW ORLEANS

Almost all loss of teeth results from either caries or periodontoclasia. These two diseases can be prevented by the necessary personal oral hygiene. They cannot be

*Studies promoted by facilities to which the author has had access at the School of Medicine, Tulane University of Louisiana, and by aid for equipment and supplies provided by the University.

†The substance of this paper was presented before a special meeting of the New Orleans Dental Association March 11, 1948.

prevented in any other way now known. The purpose of this paper is to present the oral hygiene procedure every person must follow in order to entirely prevent these diseases and their consequences, and in order to maintain the state of oral cleanliness most people would like to maintain. The personal oral hygiene procedure here presented as essential has evolved from practical application of already well known fundamental information and more recent additional pertinent information that has been published or is in process of publication.[1-4] By intensive microscopic study of extracted teeth, employing technical procedures[1, 2, 5] not usually employed for this purpose, it has been possible to secure more accurate information regarding the conditions at the locations and in the environment where caries and periodontoclasia begin.

To prevent the occurrence and progress of the lesions of these diseases their early stage must be prevented. The oral hygiene necessary to prevent these diseases, therefore, must effectively meet and counteract the etiological conditions at the locations where the lesions originate.

WHERE CARIES BEGINS

Enamel caries begins principally at or about occlusal pits and fissures and at or about the contact area between teeth. The earliest lesion consists of a "white spot" of "chalky," partially decalcified enamel. If the conditions are prolonged the lesion extends in area and depth and finally this fragile, partially decalcified enamel breaks down producing a cavity — the advanced stage of caries. The cavity, if large enough, usually can be diagnosed by the dentist but most of the earlier stage lesions cannot be recognized, except upon extracted teeth.

Some idea of the frequency and extent of these early stage lesions can be gained by very simple procedure, even without any microscopic laboratory equipment or experience. All that is necessary is to place extracted teeth (preferably from persons under 20 years of age) in 10 per cent hydrochloric acid (water 85, formalin 5, HCl 10) for one minute, then wash and brush with an ordinary toothbrush to remove the loosened cuticle, bacterial film and debris. Any "white spot," early stage caries lesions present can be seen satisfactorily (Figure 1) with the unaided eye. They contrast well with the more transparent normal enamel. The contrast is even sharper after the specimen has been allowed to dry. Under magnification, the lesions may be observed and studied better. Ordinary hand lenses are quite helpful. The dissecting microscope is still more helpful in studying such preparations.

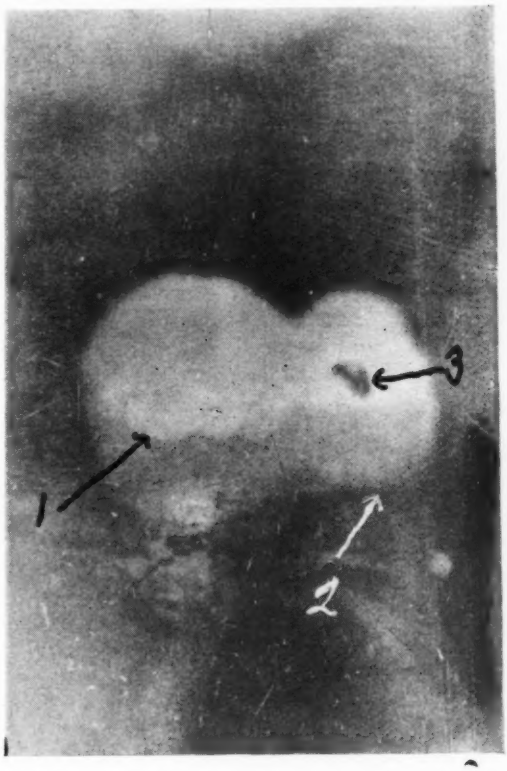


Fig. 1. Tooth from which the cuticle and bacterial film were removed by application of acid and then brushing. Note area of "white spot" (1) chalky enamel (early stage caries) contrasts with normal enamel. Cemento enamel junction (2). Small cavity (3).

It will be observed that some of these partially decalcified areas have more or less brown stain. In most instances these are old lesions which have been inactive for some time, due to changes in the environ-

mental conditions which formerly initiated the lesion and promoted activity. A good example is proximal lesions on a tooth where the contacting tooth was lost some time previously. Such inactive lesions are more often found on teeth from people past 25 years of age.

Sometimes a small broken down area (cavity) may be observed in a larger area of partially decalcified enamel which still holds its form. It will be observed that most small to medium size cavities have more or less unbroken chalky enamel about them. (Figures 2, 3).

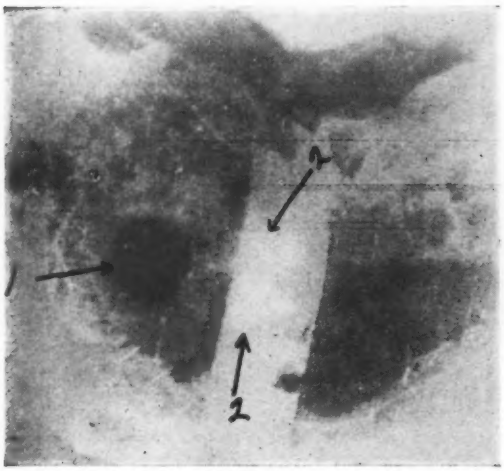


Fig. 2 Area on proximal of molar with cavity (1) and chalky enamel (2). Two parallel cuts were made through the cuticle to include the edge of the chalky enamel. Specimen in acid I minute, rinsed and then stained with crystal violet. Strip of loose cuticle removed, exposing some of "white spot" area. Clear strip from which cuticle was removed contrasts well with stained cuticle and bacterial film still in place on either side.

No cavity ever forms except as a result of breaking down of this earlier stage decalcified enamel. The early stage, partial decalcification, therefore always precedes cavity formation. Prevention of cavity formation and its consequences can be accomplished only by recognition of the etiological conditions at the location where the earlier partial decalcification occurs and by application there of effective measures for preventing or minimizing those conditions.

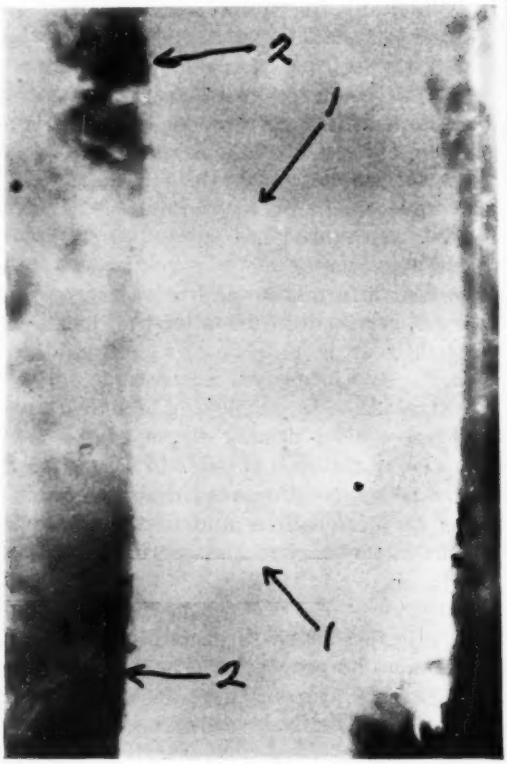


Fig. 3. Higher magnification of area on Fig. 10, showing chalky enamel extending outward from beneath cuticle on left side which was retained in place.

THE ENAMEL CUTICLE IN RELATION TO THE EARLY STAGE OF CARIES

It has been shown[2] that the enamel cuticle bears an important relationship to the early stage of caries. The enamel cuticle is an extremely thin keratin-like, transparent membrane covering the entire enamel surface at all times. It is thinner over areas where it is repeatedly worn by functional or other friction than in other areas where it is not exposed to such friction. However it is extremely thin in such areas also. In view of some confusion and conflicting opinion as to the continued presence throughout life of an enamel cuticle, it may be worthwhile to give here a simple procedure whereby anyone who is interested can clarify the matter for himself. Again this can be done without the aid of microscopic laboratory equipment or experience.

Place a tooth specimen in the 10 per cent

HCl for one minute; remove gently and dip in water for a moment to reduce the acid; place in 0.5 per cent crystal violet solution (crystal violet 0.5 gm in water 100 cc) for one minute or less; again dip in water to remove excess of stain. Now observe the loosened cuticle with the tooth immersed in a shallow dish of water in which the membrane may be teased off with some suitable delicate instrument (No. 2 or No. 7 Clevdent or S. S. White Explorer) and manipulated in the water. The cuticle itself is slightly stained and the bacterial film upon it is heavily stained. When floating in the water the membranous nature of this material from the surface of the enamel is readily recognized. One who examines a few specimens in this simple way knows, of his own knowledge, that an enamel cuticle is continuously present on teeth. This enamel cuticle is of interest in relation to caries because the bacterial film over the early stage lesion is firmly attached to the cuticle (Figure 4) and because the acid or

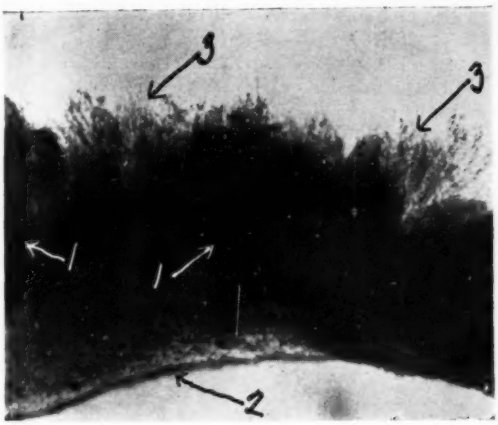


Fig. 4. Section through cuticle and attached bacterial film removed from over early stage caries. Note parallel arrangement of organisms (1) extending from cuticle (2) outward toward surface of the pile (3). Acids produced at the surface are carried as if by a wick or sponge through the material down to the cuticle.

acids which cause the first partial decalcification there must pass through the intact membrane to reach the enamel.

NATURE OF THE BACTERIAL FILM OVER THE EARLY STAGE LESIONS

A film or pad of soft bacterial material of variable thickness is present on the surface of the tooth at all areas where it is protected from removal by functional or other friction. It is thickest where it is best protected. Microscopic examination of appropriate preparations of this material ("soft tartar") shows it to be composed entirely of bacteria, usually of many different kinds. (Figure 5). One character-

Fig. 5. Bacterial film material from over early stage caries lesion torn apart, stained, mounted in 50 per cent glycerin and photographed by transmitted light, shows filamentous nature of material.

istic of such bacterial material over early stage caries lesions (and elsewhere in most cases) is that it consists mostly of long rod and filamentous forms, one end of which is attached to the cuticle on the tooth. The rods and filaments extend outward, more or less parallel to each other, toward the surface of the pile or pad. At the surface there are the growing ends and fruiting heads of the long forms of which the deeper part of the film is composed (Figures 7, 8, 9); and among these, large numbers of other bacteria of many different kinds.

PRODUCTION OF ACIDS AT CARIES LOCATIONS

Food, as it is masticated, is thoroughly and heavily inoculated with many different kinds of bacteria in the saliva, derived from all the different locations within the mouth. The bacteria in such heavily inoculated food

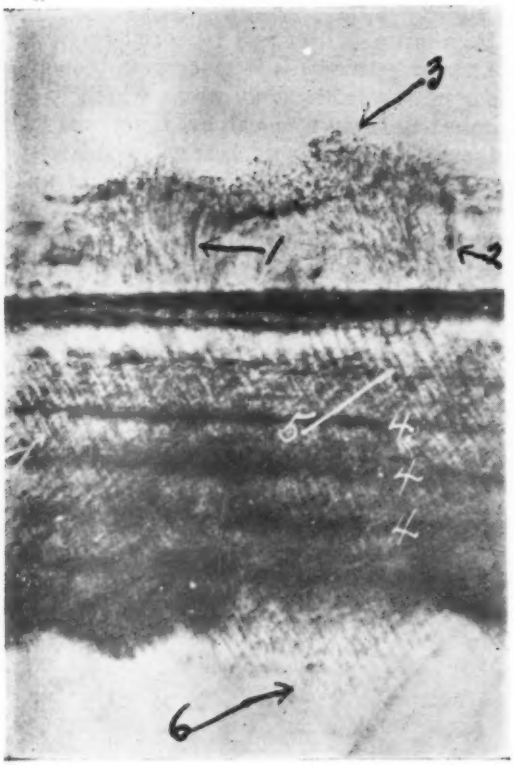


Fig. 6. Ground section, unstained, through early stage caries showing bacterial film attached to surface of tooth and indicating parallel rod and filamentous organisms (1, 2) with growing ends and fruiting heads (3) at the surface of the pile. Photographed by transmitted light which causes the white chalky enamel to appear dark. Note lines of Retzius (4) enamel prisms (5) and feathery edge at deeper part (6).

material lodged and retained upon the constantly present bacterial film pad at favorable locations about teeth, multiply and, through the action of their enzymes, break down the material which serves as their culture media. Many bacteria, when growing in the presence of favorable carbohydrates, produce acids. Such acids produced by bacteria at the surface of the bacterial film or within it, are carried, as if by a sponge or wick, through the film to the cuticle through which they pass to the enamel beneath. If such acids are produced at the particular location in sufficient strength and over sufficient length of time, decalcification of the enamel occurs. This decalcification is only partial, giving rise to the softened "white spot" caries lesion. The enamel is not completely dissolved by such weak acids in the same way as it may be, experimentally, by stronger acids. For instance, the enamel is completely dissolved and disappears (except for some remaining enamel matrix material) from a tooth immersed in 10 per cent HCl for an hour or two. On the other hand the enamel on a similar tooth immersed in 0.15 per cent HCl, even for much longer time, is not dissolved in the same way. Only partial decalcification occurs, similar to the partial decalcification of the "whit spot" caries. The enamel on the tooth becomes softer and may be broken up or crushed like a piece of chalk in much the same way as that of the early stage lesion may be. Anyone interested can confirm this observation by the simple procedure of immersing (suspending) tooth specimens in solutions of acid of the suggested strengths, for several hours (several days for weak acids).

What seems to be the same kind of partial decalcification may be produced experimentally by solutions of organic acids, such, for instance, as lactic acid—the one gener-

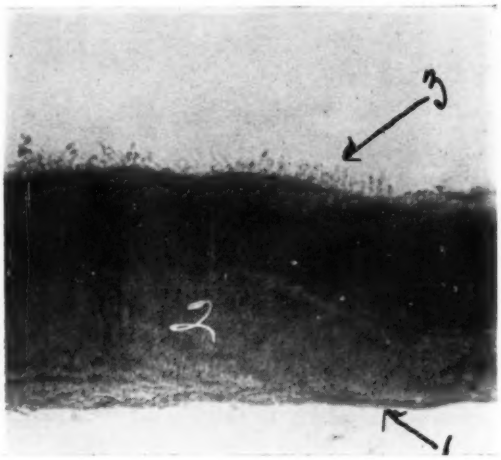


Fig. 7. Section cut through compact bacterial film pad removed from over white spot caries lesion. Note rods and filaments extending outward (2) from the cuticle (1) to which they are firmly attached and (3) fruiting heads of Leptothrix racemosa projecting at the surface.

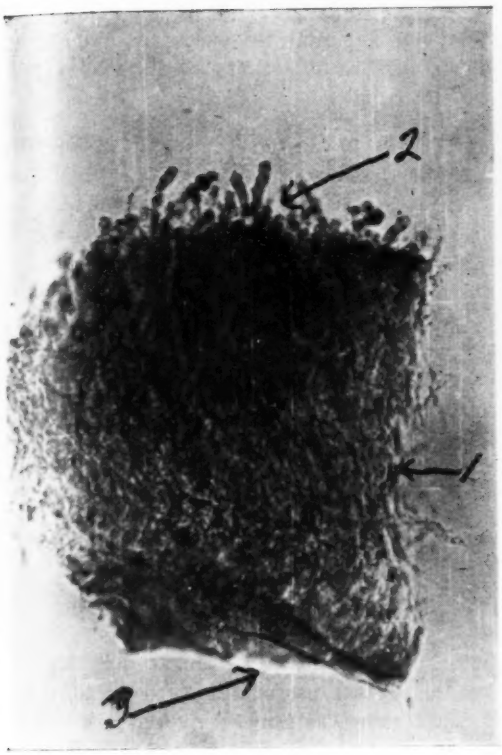


Fig. 8. Piece of the bacterial film from over early stage caries lesion. Specimen teased off, stained very lightly with safranin and mounted in 50 per cent glycerin for photographing by transmitted light. Note filamentous nature of bacterial material (1) and growing ends and fruiting heads at outer surface (2). Inner surface (3).

ally supposed to be the most important in caries production. However, weak solutions of organic acids, such as can be assumed to be formed at caries disposed locations, act very slowly. It is only after long continued exposure to such weak acids experimentally that demonstrable partial decalcification occurs. Likewise it is only after long continued and repeated production of acids by bacterial action, that a caries lesion results. It is very apparent that to prevent early stage caries at any vulnerable place on a tooth, it is necessary to prevent the formation of acids by bacteria growing there*

TIME FACTOR IN PRODUCTION OF ACIDS BY BACTERIA

Some bacteria grow and produce acids much more rapidly than others. However

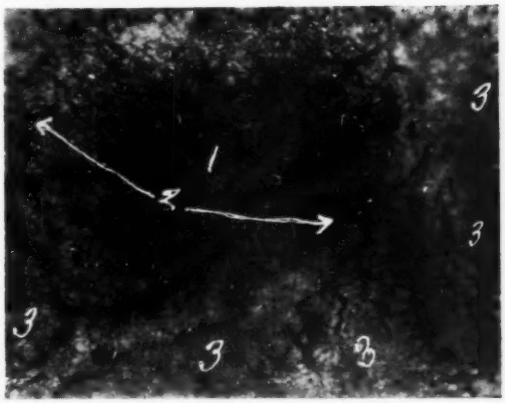


Fig. 9. Bacterial film pile on surface of tooth over early stage caries (chalky enamel) stained lightly with safranin and photographed with incident light. Note mound of *L. racemosa* (1) with some fruiting heads focused around periphery (2). Many more just out of focus.

any of them require considerable growing time, under the most favorable conditions, for production of appreciable amounts (or strengths). Perhaps the most familiar example for those who have been medical or dental students is the testing of acid production by pure cultures of different bacteria inoculated into litmus milk. Litmus milk is blue. A tube of such culture media inoculated with a loopful of a pure culture of an acid producing organism and placed in the incubator still remains blue for several hours, usually twelve or fifteen or more. After the necessary incubation time it will be noted that the color is changing to a faint pink and finally, within twenty-four hours or a longer period, to red, indicating production of considerable lactic acid from the sugar in the milk.

Stephan and Miller[15] showed that brushing the teeth thoroughly before rinsing with 10 per cent glucose solution eliminated the

*Note: I am aware of the suggestion, belief or claim by recent authors (6, 7, 8, 9, 10, 11, 12, 13, 14) that caries is, to some extent, a proteolytic process by which the organic material of the enamel is invaded and broken down by the enzymes of proteolytic bacteria and that this is followed or accompanied by disintegration or decalcification of the inorganic material. The personal oral hygiene for prevention herein specified would be equally applicable to such order of events in the caries process.

fall in pH which otherwise would have occurred. They found that in testing plaque material *in situ* for pH drop following the glucose rinse, subjects who refrained from brushing their teeth for three or four days, thereby insuring a sufficient amount of bacterial material on accessible surfaces, gave most satisfactory results. Therefore a considerable period of time is required for sufficient growth and accumulation of bacteria to occur before much acids can be produced in the presence of carbohydrates.

However another factor plays an important role in the production of acid, i. e., the amount of inoculum or number of bacteria with which the culture medium is inoculated. If, for instance, a tube of litmus milk is inoculated with say 2 or 3 cc. of rich culture, containing enormous numbers of viable bacteria, instead of the loopful as suggested above, containing relatively only a fraction as many, then we find acidity developing and the color changing within a shorter time, sometimes only a few hours.

Applying the above elementary information to our problem of preventing acid formation and thereby preventing caries, it is evident that removing from about a tooth all food material which may serve as culture media for acid producing bacteria and removing from the same location most of the bacteria, there will be little growth of bacteria (for lack of culture media) and no production of acid (for lack of fermentable carbohydrates) until food is again lodged at the particular place and sufficient time elapses for bacteria to multiply and produce acid.

Decomposing food material that has been retained and has accumulated about the teeth during the daytime gives the bacteria growing there a good start towards acid production by bedtime. If these conditions are allowed to continue through the night during sleep the most favorable conditions exist for more rapid bacterial growth and production of acids, and their action upon the teeth. As a matter of fact, the caries process progresses principally at night and during sleep. Therefore to prevent the initiation and further progress of caries the teeth must be effectively cleaned of food and accumulated bacterial material at night before retiring. Nothing else will suffice.

EARLY STAGE PERIODONTOCLASIA

Periodontoclasia begins and progresses as a local microscopic disease process. The earliest stage lesions are too small and inaccessible to be recognized except by microscopic examination of suitable sections or other preparations of the tissues involved. What is ordinarily diagnosed clinically as periodontoclasia (pyorrhaea) on the basis of flow of visible amounts of pus, receded periodontal soft tissue, alveolar resorption, pocket formation, loosened drifting teeth, etc., represents, in fact, the far advanced stage and results of a disease that usually has existed and progressed at the particular location for many years. Long previously, there was an earlier stage, actually a beginning, of the same disease about the same tooth. It is this earlier stage against which effective prevention must be directed. Measures for this purpose must be based upon clear understanding of the etiological conditions at the locations where the disease starts and from which the lesions about each tooth advance.

RELATION OF THE ENAMEL CUTICLE TO EARLY STAGE LESIONS

It has been shown[2] that the enamel cuticle bears an important relationship to the early stage of the periodontoclasia lesion. The marginal gingiva normally rests upon the smooth, non-irritating enamel cuticle. Bacteria allowed to grow and accumulate for a long time on the tooth at the gingival margin, tend, in time, to produce microscopic roughness and hardened concretion upon the cuticle. The tendency is for this to increase, not only encroaching upon the gingival margin but extending into the gingival crevices (Figure 10). In time a narrow portion of the free gingiva (the free gingiva is that portion extending occlusalward from the level of the bottom of the crevice) rests against a surface covered with hard concretion and a pack of soft bacterial material. The irritation caused by the presence of this foreign material upon the tooth, where normally smooth,

Fig. 10. Proximal of extracted tooth stained with crystal violet showing biconcave disc of bacterial material surrounding contact area, and heavy film which extended from the gingival border (gingival crevice) occlusalward. Contact point (1). Disc of bacterial film material surrounding contact point (2). Heavy bacterial material on area protected from functional friction (3). Epithelial cells remaining attached to tooth (4). Location of cemento-enamel junction (5). Some epithelial attachment tissue retained on tooth (6).

non-irritating cuticle exists, soon causes inflammation; at first only microscopic in extent. Minute and microscopic quantities of inflammatory exudate and pus cells are poured out through the inflamed tissue into the gingival crevice, and these tend to promote the growth of microorganisms and increase of the concretion. As the foreign material on the tooth increases and advances further into the gingival crevice, the soft tissues attached to the tooth are forced back by the accompanying inflammation and ulceration. Gradually and almost imperceptibly a larger and larger portion of the tooth is exposed as the gingival margin moves further away from the occlusal level. The gum "recedes."

Many factors influence the progress of the lesions at different locations about a given tooth, about different teeth in the same individual and in different individuals. For our present purpose of prevention of the disease (lesions) and prevention of further progress of lesions that already exist, it is only necessary to adopt and direct effective measures against the conditions at and within the gingival crevice. The disease process involving the more remote tissues — periodontal membrane, alveolar bone — rapidly subsides as soon as the local lesions consisting of inflamed, suppurating and broken surface of the epithelial tissue of the gum within the crevice, subside and disappear.

From the earliest stage and continuously as the lesion progresses, the tooth surrounded by this concretion and bacterial material adhering to it within the gingival crevice is, in effect, a foreign body infected with many different kinds of bacteria. It is a suppurating lesion constantly exposed to invasion by any and all of the many different kinds of bacteria within the mouth which are capable of growing in such an environment.

This foreign body effect was recognized and emphasized as the cause of periodontoclasia more than seventy years ago by Dr. John W. Riggs.[16] He supported his claims by the marked benefit and control of the disease he secured by removing this foreign material from the surface of the tooth and polishing it so it no longer caused the constant irritation and suppuration characteristic of the disease. The treatment had to be repeated frequently as the material soon reformed. How different it would have been if he and those who followed him could have had their patients carry out the personal oral hygiene procedure we now know to be possible and essential, thereby preventing the recurrence of the local conditions which originally caused and promoted the disease!

MATERIAL UPON THE TOOTH AND WITHIN THE CREVICE

In order to devise and adapt personal oral hygiene measures for prevention of the formation of the material upon the tooth which causes and promotes progress of the disease, correct conception of the nature of this material is necessary. We have already seen that the bacterial film (tartar) on the surface of a tooth above the gingival margin at the more protected places where it can accumulate and where it is not dislodged by functional friction (Figures 10, 11), consists largely of a thick pad or pile of long rod and filamentous bacteria, one end of which is attached to the enamel cuticle. The other end extends outward to the surfce of the film pack where there are usually an abundance of other bacteria of many different kinds.

Fig. 11. Proximal of extracted molar stained to show bacterial film. Contact area (1). Heavy bacterial film (2). Epithelial cells remaining attached to tooth (3). Location of cemento-enamel junction (4).

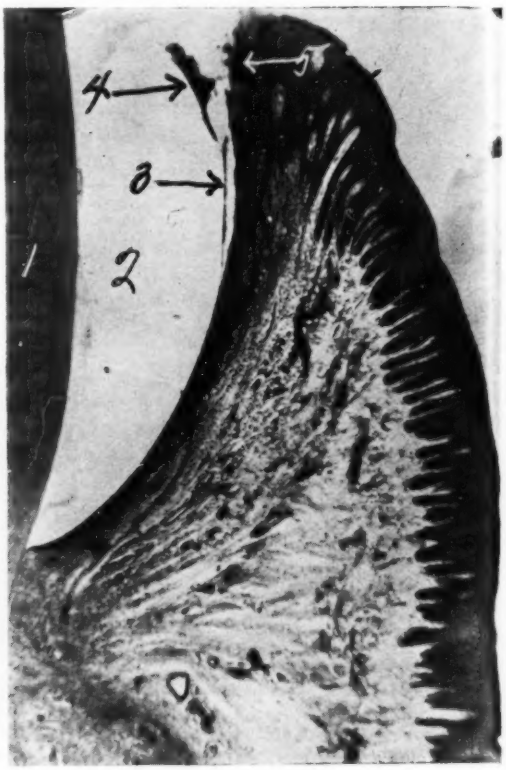


Fig. 12. Section through gingival tissue showing foreign material attached to cuticle within the crevice. The early stage of periodontoclasia. Dentin (1). Enamel space (2). Cuticle (3). Bacterial film and concretion on cuticle against which the inflamed free gingiva (5) must rest.

The gingival margin must rest against this bacterial mass of foreign material which causes irritation, inflammation and suppuration. As the foreign material on the tooth builds up and advances into the gingival crevice (Figure 12) the inflammatory exudate there offers favorable environmental conditions and nutritive material for the growth of other types of microorganisms that do not grow outside of the crevice. They are organisms, such as certain leptotrichia, actinomyces, spirochetes, ameba, etc., which prefer or require the more or less anaerobic conditions, inflammatory tissue exudate, blood and pus present in such diseased gingival crevices. After a lesion, although small, is well established at any particular location and any time thereafter during the advancement of the lesion, the surface of the tooth within the crevice against which the inflamed gingival surface rests, has more or less hard calculus on it at most areas. The inner border of such calculus approaches but usually does not quite reach the zone of disintegrating epithelial attachment cuticle,[1] a landmark that can be seen on extracted teeth and indicates the exact location of the outer border of the epithelial attachment. Superimposed upon and attached to the calculus and any part of the tooth on

which there is none, within the gingival crevice, there is a pad or pile of soft bacterial material. This bacterial film consists largely of closely packed parallel long rod and filamentous forms, one end of which is attached to the calculus or the tooth from which the rod or filament extends outward toward the surface of the pad against which the inner inflamed surface of the gum rests. At the surface of the bacterial film within the crevice there are growing ends and fruiting heads of the rods and filaments composing the pad, and among these more or less other bacteria that invade the lesion from the mouth. Among elements making up the film pad on the tooth within the gingival crevice perhaps the stems and fruiting heads of *Leptothrix falciformis* are the most noticeable and the most constant. This organism was first described in material from around teeth by Beust[17] in 1906 and 1908. I[5] have called attention to the fact that the habitat of *Endameba buccalis* is among the stems, branches and fruiting heads composing this film pack.

We do not know exactly what role any of the different microorganisms found in the gingival crevice play relative to disease there. Probably it is a very complex process. However it is clear that for the purpose of preventing initiation of the lesions of this disease and preventing further progress of lesions that have already been established, effective measures must be applied to prevent or minimize the growth and accumulation of bacterial material on the tooth at the entrance to, and within, the gingival crevice, which cause the disease (Figures 10, 11, 12).

Food material retained at the entrance to the gingival crevices and between the teeth, and packed into the crevices, promotes growth of bacteria there and increases inflammation of the tissues against which it rests. If food and bacterial material that has accumulated at and within the gingival crevices during the day is effectively removed at night before retiring, there follows a period of many hours during which there is greatly lessened bacterial growth. Rapid subsidence of inflammation occurs. Therefore to effectively prevent the initiation and further progress of periodontoclasia lesions the teeth must be cleaned at and within the gingival crevices every night before retiring. Nothing else will suffice.

DIAGNOSIS OF EARLY STAGE PERIODONTOCLASIA

The periodontoclasia lesion begins at the entrance to and just within the gingival crevice, principally the interproximal crevices, the distal and mesial crevices about teeth where there is no approximating tooth and the crevices at the buccal, labial and lingual embrasures. At first the lesion is only microscopic in extent. Even then there are a few (often only microscopic quantities) pus cells passing through the inflamed inner surface of the free gingiva. These tend to accumulate just within the crevice and at the entrance to it. Material removed from within the crevice by proper technic, stained and examined shows some to many pus cells. There are no pus cells within uninflamed healthy gingival crevices. Therefore, the presence of pus in material from the gingival crevice is diagnostic of inflammation there. Likewise the absence of pus cells means absence of inflammation and absence of periodontoclasia.

Microscopic examination for pus is a very simple procedure. Material must be properly taken from the gingival crevice, spread upon a microscope slide and stained with some one of the many appropriate stains for such specimens. The material must be delicately scraped from within the crevice with an appropriate small instrument. Although some of the instruments usually used by dentists for other purposes may be used, the best results can be obtained with an explorer and scraper which the author first made from a D. C., R & L, Premierlite explorer (Figure 13). The blade is ground to a width of .55 mm. and suitably shaped for entrance and manipulation within the gingival crevices—especially the interproximal crevices. A cross section of the blade is half disc shaped. With it one can obtain small amounts of

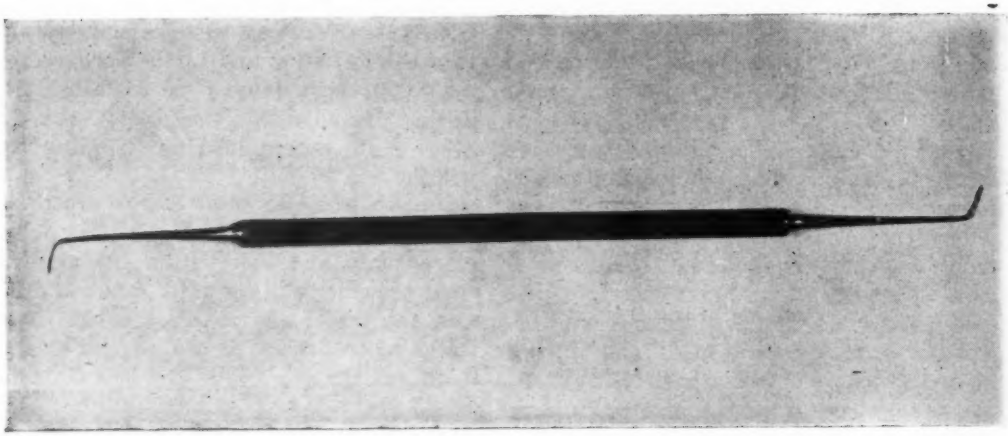


Fig. 13. Explorer and scraper ("Bass' 55") for exploring, scraping and securing material from gingival crevices and periodentoclasia lesins.

material from the inflamed surface of the gum within the crevice and from the very bottom of the space, where the disease is progressing. Success depends upon correct technic in collecting and preparing material for examination rather than upon collecting large amounts improperly.

The material is spread in a small area (2 to 5 mm. in diameter is enough) upon a microscope slide. Removal of the small amount of material from the blade of the explorer and spreading it upon the slide can be facilitated by the use of some kind of teasing needle or other small pointed instrument. Several such specimens from different crevices can be placed at different locations on one slide, and all stained and examined at the same time. The author usually mounts on one slide for examination all the specimens taken at a sitting from a given subject.

The slide is ready for staining and examination. Any one of many staining methods may be used satisfactorily. A good one for general purposes is as follows: (1) Fix with heat; (2) Carbol fuchsin one-half minute; (3) Rinse; (4) Crystal violet one-half to one minute; (5) Wash, dry and mount in oil for examination.

Pus and other cells are satisfactorily seen with the low power (16 mm.) objective. Higher powers and the oil immersion objective are needed for study of the bacteria, spirochetes and ameba *Endameba buccalis*) that may be present.

WIDESPREAD PREVALENCE OF PERIODONTOCLASIA

Periodontoclasia is practically a universal disease. There are suppurating lesions about some or all of the teeth of all people except those who have learned and follow the necessary personal oral hygiene to prevent the disease. Anyone who is interested can confirm this by examining, as indicated above, material from his own interproximal gingival crevices, and from those of others.

Inflamed and ulcerated inner surfaces of the free gingiva bleed easily from the slightest force or manipulation. Uninflamed epithelial surfaces do not bleed Therefore "bleeding gums" can be considered practically diagnostic of inflammation and ulceration—the early stage of periodontoclasia.

In view of the widespread prevalence of this disease it is hardly necessary, for diagnostic purposes, to make microscopic examinations for pyorrhaea, as suggested above. It can be assumed to be present about some of the teeth of practically all adults and most younger people. During a period of several years the author has examined a considerable number of people, mostly medical students and other university personnel. In no instance has he failed to find pus from some of the gingival crevices, and also one or more demonstrable lesions where some receding of the gingival

attachment has taken place. This experience, which will be confirmed by those who employ appropriate technic, indicates the extensive prevalence of the disease, the inadequacy of the oral hygiene procedures presently in general use and the need for a better method.

NECESSARY PROCEDURE

We may now state the fundamental facts to which the necessary oral hygiene procedure must conform and specify the procedure required. One of these facts is the time honored saying "a clean tooth does not decay." The other, more recent, is "periodontoclasia does not occur about a clean tooth"[8] The author has formulated one sentence which comprehends what every person must know and do to save his teeth from these diseases and to maintain reasonable personal oral cleanliness. It is used as a slogan in teaching personal oral hygiene to others. Anyone who undertakes to teach others how to take care of their teeth (after learning how to take care of his own) will find this sentence of instruction useful and helpful. *"You must clean your teeth right with the right kind of both toothbrush and dental floss every night before retiring."* No part of this sentence may be changed or omitted without impairing its completeness. In the light of present information, no part of these instructions may be disregarded or neglected by anyone except at the jeopardy of his dental heatlh.

All other supposed preventive measures which conflict with, or are intended to supplement, what is comprehended in the above sentence, tend to confuse or detract from the personal oral hygiene that is essential for maintaining oral health and cleanliness. If the teeth are also cleaned partially or well at other times, this contributes to greater oral cleanliness, but under no cirmustances may such cleaning at other times of the day take the place of the essential cleaning at night before retiring.

Heretofore the individual has not known exactly how to clean his teeth right and the right kind of toothbrush and dental floss have not been available. Therefore he must be taught by someone who does know.

RIGHT KIND OF TOOTHBRUSH

The function of the toothbrush is to dislodge and remove from any and all areas on the teeth that are accesible to the application of the bristles of the brush as much as possible of the decomposing food and bacterial material that has accumulated and is retained there since the previous cleaning. This material is soft, often microscopic in amount and composed of microscopic particles (bacteria and food elements). Its presence and character can be ascertained only by appropriate microscopic examination. The most important places to be cleaned with the brush are (a) the occlusal pits and fissures, (b) the proximal surfaces in the sulci between teeth as far as the bristles may go and (c) the surfaces of the teeth within the gingival crevices wherever they are accessible to the application of the bristles of the brush.

Material is dislodged and removed by the digging action of the ends of the bristles when the brush is applied firmly against the places to be cleaned and moved back and forth with short strokes ("vibratory motion"). The brush must be pressed down hard enough to force some of the bristles into the pits, fissures, sulci and gingival crevices as far as their diameter will allow them to go. The bristles must be flexible enough to allow those that do not enter the deeper spaces at the moment, to be deflected and not prevent others from entering. They also must be flexible enough so they bend and do not injure the gingival tissue when applied directly to the gingival crevices, and manipulated so as to secure the necessary digging action to dislodge the foreign material on the tooth within the crevice. For the same reason the ends of the bristles must be round and smooth instead of sharp, jagged, chisel shaped and rough, as the bristles of so many current toothbrushes are (Figure 14). The shape, size and form of the brush must be such as to adapt it to the most practical and effective application and manipulation for the purpose for which it is used (Figure 15).

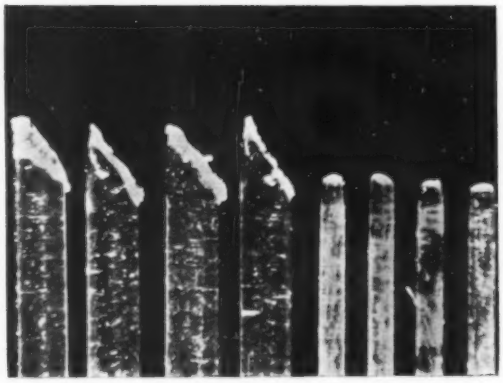


Fig. 14. Selected .014″ bristles (left) from current toothbrush by the side of .007″ bristles with smoothed and rounded ends from right kind brush.

The specifications for the right kind of toothbrush are:

1. Plain straight-handle design; over-all length about 6″, width about 7/16″; 3 rows of bristles, 6 tufts to the row, evenly spaced (Figure 16).

2. High quality nylon bristles, about 80 per tuft, .007″ diameter, straight trim, finished to 13/32″ length.

3. Ends of bristles ground and finished to hemispherial shape or at least so as to eliminate all sharp points and rough edges.

4. A similar brush of reduced size for the use of young children should have an over-all length of about 5″, .005″ bristles, finished to 11/32″ length.

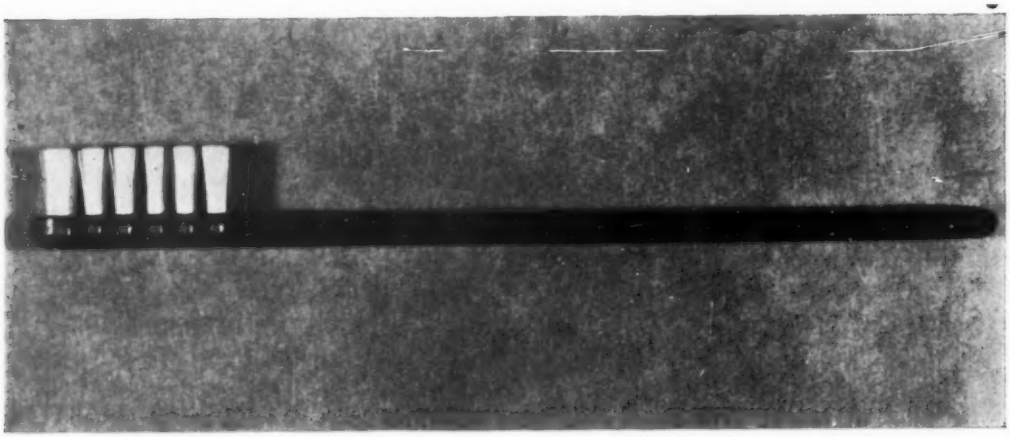


Fig. 15. Right kind tootbrush referred to in this paper and described elsewhere (3). Note straight handle and construction and straight trim.

Based upon much study of the spaces to be cleaned, the character of the material to be removed, and the conditions to be met, the author has specified elsewhere[3] the optimum characteristic of toothbrushes for personal oral hygiene, giving the reason for each of the characteristics specified. The specifications laid down meet the requirements indicated above. This, and no other, is the right kind of toothbrush. In the light of present information any brush that deviates from the characteristics specified is less effective and less appropriate for the purpose, to the extent it so deviates.

BRUSHING THE TEETH

All the surfaces of all the teeth to which the brush can be applied, should be brushed. A good system is to brush the buccal and labial surfaces of all teeth first, then the occlusal and lingual surfaces of the grinders in all four quadrants and finally the lingual surfaces of the anterior teeth. The bristles of the heel of the brush can be applied most effectively to the lingual surfaces and the gingival crevices of these latter teeth. The bristles of the distal end or toe of this right kind of brush can be applied most effectively back of the last tooth in each quadrant by tilting the brush for

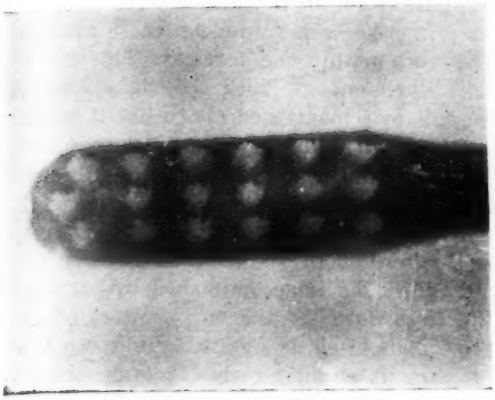


Fig. 16. Face view of right kind toothbrush, showing distribution of tufts.

this purpose at the same time the occlusal and lingual surfaces of the grinders are brushed. Anyone should be able to brush all of these teeth well enough for all purposes in less than one minute.

DENTIFRICES

The question of dentrifices necessarily arises. If one's hands are soiled with food and other objectionable material, he washes them with soap and water. A touch of soap (toilet soap) on the brush helps to clean similar material from the teeth. Nothing else is necessary for routine purposes.

The teeth of many people become stained with various substances such at tobacco, tar, certain stains in food and beverages, sometimes stains produced by chromogenic bacteria. Such stains are retained by the bacterial film but do not pass through it into the tooth. They may be removed and minimized by a mildly abrasive powder on the brush. Ordinary prepared chalk is effective. When used with the right kind of brush here suggested, it is harmless. It may be used as frequently as the individual requires. The teeth of some individuals stain much worse and in shorter time than others. Each person should use prepared chalk as often as necessary to prevent objectionable discoloration of his teeth. Some will require it every day, others only once in several days or longer.

The sweetening and strong mint or other flavors which most dentifrices contain serve no useful purpose and are more or less harmful.

CLEANING THE PROXIMAL SURFACES

No matter how much or what kind of brushing is done, it is not possible for the bristles to reach and clean the proximal surfaces between the teeth. It is simply imagination to think otherwise. At the contact point the teeth are in direct contact and there is no space between them. For a variable distance extending outward from the contact plane in all directions there is a gradually widening space which is filled with a pack of bacteria, mostly long rod and filamentous type. This material has the form of a somewhat irregularly outlined biconcave disc (Figure 10) with the center corresponding to the contact point. When heavily inoculated food material is lodged upon the outer part of this biconcave disc where there are large numbers of growing ends and fruiting heads of the rods and filaments of which it is composed, acids may be produced there and may be carried, as if by a sponge or wick, deeper into the space. If the acid production continues long enough, ultimately there is partial decalcification—early stage caries—and later perhaps breaking down, cavity formation—advanced stage caries. In order to surely prevent these events it is absolutely necessary to clean the proximal surfaces of the teeth in this area every night before retiring. When done right this removes most of the bacteria and the food material in which they could grow and produce acids. There is not sufficient time from the time food is put in again the next day, for maximum growth of bacteria and for production of harmful amounts of acid, before time to clean the teeth again at night before retiring.

The only way now known, and the only way likely to ever be known, whereby the bacterial film on the proximal sides of the teeth can be removed is by the proper use of the right kind of dental floss. Elsewhere[4] the author has specified the optimum characteristics of dental floss for personal oral hygiene, indicating the necessity

or basis for each characteristic specified. This right kind of dental floss consists of 170 very fine filaments of high tenacity nylon. It is not waxed, and is only slightly twisted (3 turns to the inch). When drawn across the surface of the tooth, each of the 170 separate filaments is potentially capable of mechanically dislodging and removing some part of the microscopic bacterial material thereon. Also the bundle of loosely held together filaments is capable of receiving and holding in the spaces between the filaments (Figure 17), large numbers of microscopic particles (bacteria).

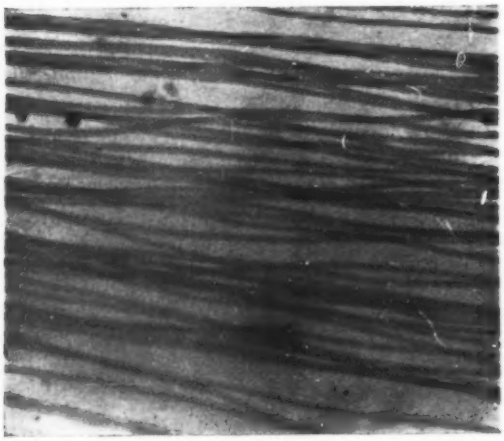


Fig. 17. Less than one-third the total number of nylon filaments contained in the right kind dental floss. Each separate filament, when properly used, is potentially capable of scraping off or dislodging some material and the many spaces between them can hold many microscopic particles, bacteria, etc.

NECESSITY FOR CLEANING THE TOOTH WITHIN THE INTERPROXIMAL GINGIVAL CREVICES

So far as proximal caries of the enamel is concerned, it is only necessary to clean the proximal surfaces of the tooth above the gingival margin or the papilla. We have seen that from the very earliest stage of periodontoclasia there is bacterial film and other foreign material on the surface of the tooth within the gingival crevice (Figure 12) and that this material is responsible for the initiation of the very earliest lesion and for the continued progress of the disease. To prevent the beginning and progress of the inflammation and suppuration which characterizes the disease it is necessary to clean these areas of the teeth within the crevices. This can be done well enough with the right kind of dental floss mentioned above but it cannot be done in any other practical way now known. The surfaces of all teeth within the interproximal crevices of contacting teeth, and those within the distal or mesial crevices where there are no contacts, must be cleaned. This is accomplished by carrying the floss down to the very bottom of the crevice, holding it against the tooth and drawing it slightly endways and outward so as to scrape the surface. The bacterial material is dislodged and much of it is held and removed within the spaces between and around the filaments (Figure 17) of the floss.

DETAILED DIRECTIONS FOR CLEANING THE TEETH RIGHT WITH THE RIGHT KIND OF DENTAL FLOSS

While different people may develop their own technic and manipulations for cleaning their teeth with dental floss, the following procedure is probably the most practical and effective:

1. Cut off a piece of floss about 2 to 3 feet long.

2. Wrap one end with 2 or 3 turns around the first phalanx of the right index finger, for the purpose of anchoring or holding it. (Figure 18).

3. Bring the floss over the end of the right thumb which is also held against the finger around which the floss is anchored. (Figure 18).

4. Grasp the floss with the left hand and bring it over the end of the first finger of that hand. Thus a length of floss, about 1 inch long, is held between the thumb of the right hand and the first finger of the left hand. (Figure 18 (1)).

5. Now with the thumb inside of the cheek and the finger inside of the mouth, the floss is carried to the very bottom of the gingival crevice back of the last right upper tooth, drawn slightly endways through the crevice and crossways outward across the distal surface so as to scrape off and dislodge the soft bacterial material on the tooth within the crevice and outwards.

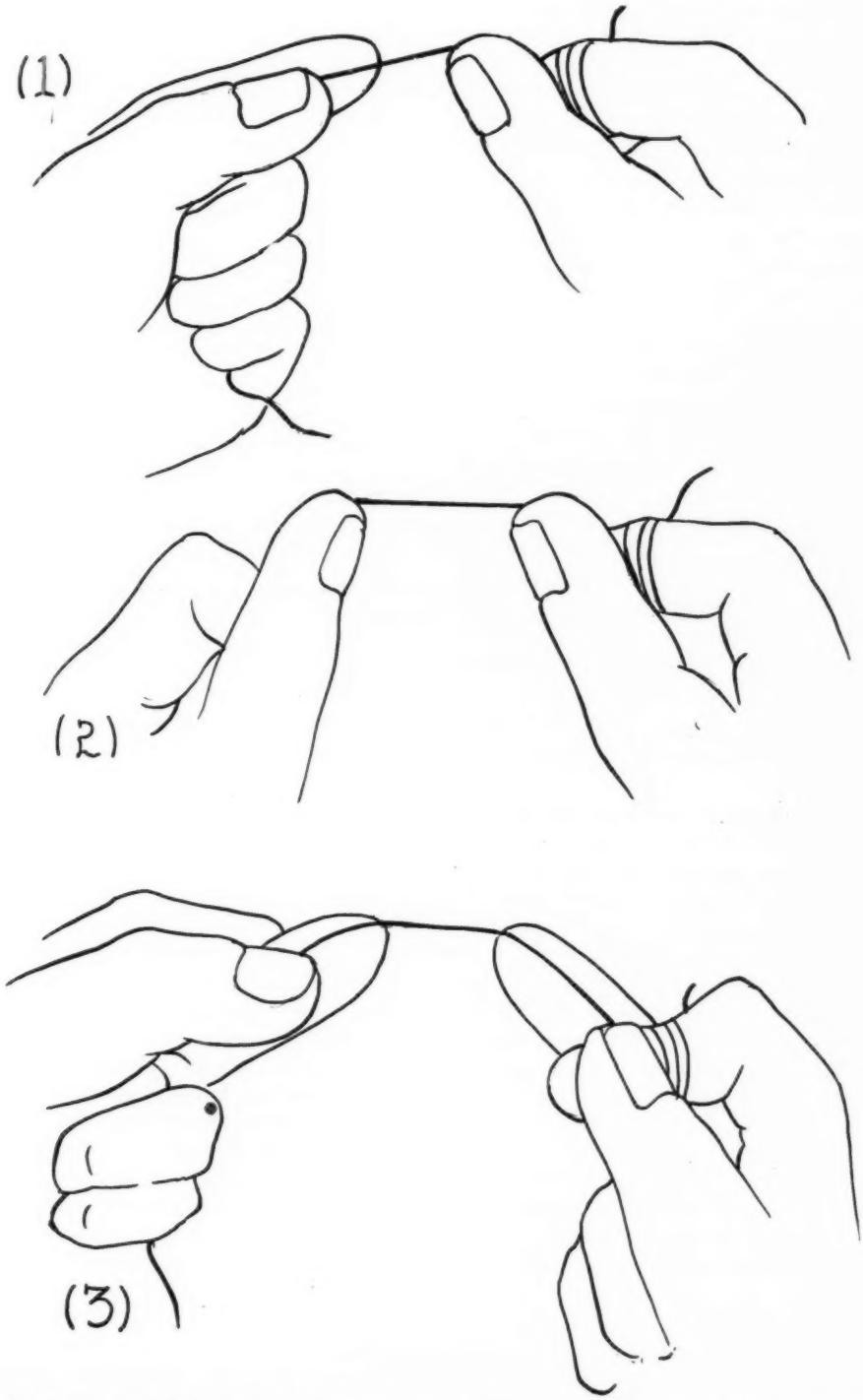


Fig. 18. Best way to hold floss in cleaning the teeth. For upper right (1). For uper left (2). For lowers (3).

6. Holding the floss in the same way, pass it into the next interproximal space. Carry it to the bottom of the posterior gingival crevice and clean the mesial surface of that tooth. Now, before withdrawing the floss from this interproximal space, clean the distal surface of the other tooth in the same way. Then withdraw the floss and move on to the next interproximal space, etc., until the proximal surfaces of all teeth have been cleaned.

7. In passing the floss between contacting teeth it is not forced directly in and out. It should be held over the contact and drawn gently and slightly back and forth endways. This allows the low-twist, unwaxed floss to flatten and pass between the contacting teeth with the greatest ease.

8. After cleaning 2 or 3 teeth the part of the floss used is somewhat soiled and loaded with bacterial material. It is desirable to move along the string to a new place by taking another turn around the anchoring finger. This should be repeated from time to time as needed.

9. The floss is held and manipulated with the same fingers as indicated above until after the surfaces of the teeth in the interproximal space between the left central and lateral have been cleaned.

10. In cleaning the rest of the upper teeth, it will now be found more convenient and practical to hold the floss over the ends of the thumb of the right hand as before and over the thumb (instead of the index finger) of the left hand. (Figure 18 (2)).

11. All the lower teeth now should be cleaned in the same way. Most people will find that they can carry out the necessary manipulations most successfully with the floss held over the ends of the second finger of each hand instead of the thumbs or the thumb and first finger as in cleaning the upper teeth. (Figure 18 (3)).

12. After cleaning all the teeth with dental floss, the mouth should be thoroughly rinsed by forcing water vigorously back and forth between the teeth in order to remove material that has been loosened or dislodged but not removed by the floss. After a little experience one can clean all his teeth well with dental floss in from 2 to 3 minutes.

13. It gives a pleasurable sensation of cleanliness to hastily brush the teeth again after cleaning them with dental floss. But this is not essential.

RESULTS

The author has instructed and had under observation, a sufficient number of subjects to be able to state positively the beneficial effects that result from the personal oral hygiene herein specified.

1. No new caries lesions develop.

2. Early stage lesions (mostly unrecognized "white spot" partially decalcified enamel that has not broken down) do not progress further or break down.

3. Small shallow cavities do not progress but usually become inactive.

4. Correctly made fillings do not undermine or break down.

5. No new periodontoclasia lesions occur.

6. All early stage periodontoclasia lesions heal promptly. It is almost dramatic the way in which the bleeding from the gingival crevices stops entirely after the first few days. Pus (even microscopic quantities) is no longer present in material from most of the crevices, and greatly diminished in that from others. The delayed or incomplete healing in such lesions is usually due either to calculus or scale on the tooth within the crevice or to irregularities or other conditions on the surface of the tooth which prevent accurate application of the floss. In most such instances, removal of the foreign material from the surface of the tooth at, and within, the gingival crevice by the dentist, followed by the right personal oral hygiene, results in prompt subsidence of the disease.

7. Each advanced stage periodontoclasia lesion and deep "pyorrhaea pocket" is a separate problem. However, cleaning off the tooth at and within the lesion by the dentist at suitable intervals together with faithful application of the personal oral hygiene described herein usually will yield most gratifying results. The beneficial results will depend largely upon the extent of the lesion and the damage already done. In favorable

instances suppuration and inflammation of periodontal tissues subside, and loose, drifting teeth usually stabilize.

8. Foul odors from the mouth due to decomposition of food material about the teeth, to putrefaction of inflammatory tissue exudates within the crevices and to the growth of certain microorganisms (especially spirochetes) in the blood enriched material in the crevices, is avoided.

9. Much satisfaction is derived from the sense of oral cleanliness which one enjoys, after he once understands the conditions and learns how to clean his teeth effectively.

COMMENT

Every person who has teeth to save and everyone who desires to maintain reasonable oral cleanliness must learn and follow the personal oral hygiene procedure herein described. People go to dentists for treatment of the advanced stage of caries and periodontoclasia from which nearly all loss of teeth results. They do not know or properly evaluate the fact that the lesions, representing more or less irreparable damage, could have been prevented. Neither do they recognize the presence of existing earlier stage lesions, further progress of which can be prevented by personal oral hygiene. They need to be instructed.

Dentists should be interested in teaching this necessary personal oral hygiene to their patients not only for the purpose of prevention but also to greatly improve the success and durability of their treatment of existing lesions and conditions. To fill a cavity without making certain, at the same time, that the patient knows how to maintain the necessary cleanliness of the area in the future reduces, on the average, the usefulness and success of the work done. To clean the accumulations of foreign material from the teeth or to treat his periodontoclasia without, at the same time, teaching the patient how to keep his teeth clean in the future, greatly reduces the value of the service. The value of the periodical visit to the dentist for check up and "prophylaxis" is very greatly increased if the patient is also taught this necessary personal oral hygiene.

It is evident that the practicing dentist should teach the necessary personal oral hygiene to his own patients. However, to teach it he must first know it himself. It is axiomatic that one cannot teach what he does not know himself. Except for anyone who may have already learned how to clean his teeth right, as here indicated, the dentist who still has teeth left now has more or less suppuration within the gingival crevices, and therefore active periodontoclasia, about some or many of his teeth. This will be confirmed by microscopic examination of properly collected material from his interproximal gingival crevices. He is losing his own teeth from the same conditions for which his patients need advice and treatment. Until he learns and practices the necessary personal oral hygiene to save his own teeth, he is not very well prepared to instruct his patients how to save theirs. Therefore he should first learn and practice the right method himself. Then he will realize how necessary it is for his patients also and can instruct them correctly and effectively.

SUMMARY

The necessary personal oral hygiene for prevention of caries and periodontoclasia has been presented in some detail. It conforms to the two fundamental facts: "a clean tooth does not decay" and "periodontoclasia does not occur about a clean tooth." The essentials are embraced in the teaching slogan which the author has formulated, uses, and recommends that others use: *You must clean your teeth right with the right kind of both toothbrush and dental floss every night before retiring.*

REFERENCES

1. Bass, C. C.: A demonstrable line on extracted teeth indicating the location of the outer border of the epithelial attachment. J. Dent. Research, 25:401. 1947.

2. Bass, C. C.: The enamel cuticle. I, in relation to the early stage of caries; II, in relation to the early stage of periodontoclasia. To be published soon.

3. Bass, C. C.: The optimum characteristics of toothbrushes for personal oral hygiene. Dent. Items Int. 70:697, 1948

4. Bass, C. C.: The optimum characteristics of dental floss for personal oral hygiene. Dent. Items Int. 70: Sept., 1948.

5. Bass, C. C.: The habitat of Endameba buccalis in the lesions of periodontoclasia. Proc. Soc. Exp. Biol. and Med., 61:9. 1947.

6. Frisbie, H. E., Nuckolls, J. and Saunders, J. B.: Distribution of the organic matrix of the enamel in the human

tooth and its relation to the histopathology of caries. J. Am. Col. Dent., 11:243. 1944.

7. Malleson, H. C.: The histology of enamel caries. Brit. D. J., 46:907. 1925.

8. Bodecker, C. F.: The bacterial invasion of the enamel in dental caries. D. Cosmos, 69:987. 1927.

9. Bodecker, C. F. and Bodecker, H. W. C.: The bacterial destruction of dental enamel. J. D. Research, 9:37. 1929.

10. Bibby, B. G.: The organic structure of dental enamel as a passive defense against caries. J. D. Research, 12: 99. 1932.

11. Berke, J. D.: Studies in the histology and pathology of human enamel. D. Cosmos, 78:700. 1936.

12. Ibid, New finding in the histology and pathology of human enamel. J. A. D. A. 27:1229. 1940.

13. Frisbie, H. E. and Nuckolls, J.: Caries of the enamel. J. D. Research, 26:181. 1947.

14. Gottlieb, B.: Dental caries. Philadelphia, Lea and Febiger. 1947.

15. Stephan, R. M. and Miller, B. F.: A quantitative method for evaluating physical and chemical agents which modify productivity of acids in bacterial plaques on human teeth. J. D. Research, 22:45. 1943.

16. Riggs, J. W.: Suppurating inflammation of the gums and absorption of the gums and alveola process. Penn. J. D. Sci. 3:99. 1876.

17. Beust, Theo. Von: A contribution to the morphology of the micro-organisms of the mouth. Dent. Cos., 50:594. 1908.

18. Bass, C. C.: Prevention of the loss of teeth. The Mississippi Doctor, 20:522. 1943.

DIAGNOSIS OF TUBERCULOSIS*

HERBERT L. MANTZ, M. D.

KANSAS CITY, MISSOURI

ADVANCES IN DIAGNOSIS AND TREATMENT

In the last decade there has been a tremendous change in the diagnosis and treatment of chest disease. Among the important advances in *diagnostic measures* are:

1. The widespread use of photofluorography and the increased use of x-ray, including section radiography, special positions, bronchography and the use of intravenous dyes.

2. There has been a better appreciation of the tuberculin and other intradermal tests.

3. Laboratory methods, especially culture technics, have been improved.

4. There has been increased use of special methods to secure sputum specimens, such as gastric lavage and bronchoscopic aspirations. The use of the bronchoscope as a diagnostic tool has become quite common.

5. There is much more knowledge of fungus infections in the lungs.

The principle advances in *therapy* have been:

1. The discovery and use of the antibiotics and the fact that the sulfa group, penicillin and streptomycin, are especially valuable in so many pulmonary infections.

2. Tremendous advances in chest surgery have been made in both tuberculous and in non-tuberculous disease. Resections for tumor, bronchiectasis and tuberculosis are rapidly increasing.

These advances have occurred concurrently. Most of the therapy improvements would be impossible without similar advances in diagnostic knowledge. The sum total of all of these results in more specific diagnosis and treatment. We formerly talked in anatomical terms, but with the development of specific treatment we must have specific etiological diagnosis. When the treatment of tuberculosis was for the most part bed rest, it was not likely to be fatal for some non-tuberculous patients to receive such treatment, but when we use such permanent methods as lung resection or thoracoplasty we must be sure that our diagnosis is correct. Also, there was a time when it made little difference whether or not a correct diagnosis was made for bronchiectasis or lung cancer. Now, however, with specific treatment for these diseases available, it is very necessary that they be diagnosed accurately and as soon as possible if therapy is to be successful. Also, it is a very grave injustice to label a patient as being tuberculous when he is not. Therefore, it is very important that when a positive diagnosis of tuberculosis is made, that it be correct.

The widespread use of the photofluorographic unit in routinely x-raying large groups of supposedly healthy people has discovered many persons with significant chest findings. It is a generally accepted practice to refer such suspect-cases to family doctors for follow-up and differential diagnosis. In order to prevent mistakes and loss of time a definite routine is advisable. Another by-product of the extensive tuberculin and x-ray surveys has been the revision of our ideas of the value of history and

*Read before the Sixty-eighth Annual Meeting of the Louisiana State Medical Society, Monroe, April 14, 1948.

physical diagnosis. We know now that there is no reliable group of signs and symptoms that points absolutely to a diagnosis of tuberculosis. We have unexplained hemorrhage from tuberculosis, but also from bronchiectasis and lung tumor, not to mention heart disease and the many other causes. Pulmonary hemorrhage is practically always pathological and demands immediate and unceasing effort to find the cause, but it is not of sufficient weight per se upon which to base a tuberculosis diagnosis. The same applies to a history of pleurisy with effusion. A parenchymatous x-ray lesion is also not enough to justify a sure diagnosis, neither are persistent rales in the upper half of the chest. It is rare indeed to find rales in the upper chest without finding x-ray evidence. On the other hand, it is very common to have x-ray findings and no clinical findings. We know that patients who wait for signs and symptoms to develop before seeking medical advice usually have far-advanced disease. The careful family physician looking for tuberculosis among his patients conducts a case finding survey just as any public health agency would do, by routinely making tuberculin tests and x-ray films of the reactors. He must always think of the possibility of tuberculosis. This is the only way that minmal cases may be found with any consistent degree of success.

To establish a positive diagnosis of tuberculosis the following criteria must be considered:

1. Positive tuberculin test.
2. Positive sputum on smear or concentrate.
3. A positive gastric culture. Cultured acid fast bacilli must be proven pathogens.
4. X-ray shows a parenchymatous lesion.
5. Pleurisy with effusion is present.

TUBERCULIN TEST

The tuberculin test should be *positive*. If this test is not positive, tubercle bacilli must have been found in the sputum. The tuberculin test should be an intradermal one. The best dose seems to be .0001 mg. P.P.D. or 0.1 mg. of old tuberculin. There is a difference in strength of tuberculin as prepared by different commercial houses. Clinicians should select a tuberculin with which they are familiar or can become familiar and stick with it. We have found that purified protein derivative is fairly satisfactory and have now used it for several years. The dose of .0001 mg. is an intermediate strength which many surveys and studies have shown is probably the critical test strength for this product. To make this dilution the two tablet, second strength package is purchased and one tablet diluted with one-half "cc" of the diluent as directed. Then add the one-half "cc" to a 20 "cc" vial of buffered diluent. This makes a slightly higher dose than .0001 mg. per c.c. but not enough to make any difference. The two tablet package and the buffered diluent can be purchased in any good drug store supplying physicians so should be readily available for everyone. In our experience practically every active case of tuberculosis will react to this test. I do not believe that higher strengths are helpful. It is true that sometimes we get reactions to higher strengths but most of these are atypical and unless one is skilled in interpretation, are hard to read. A positive test with .0001 mg. must have an area of real edema at least 5 mm. in diameter and should have some erythema surrounding it. If there is no edema, the test is negative. If you feel that the test is doubtful or is a false negative, purchase new material, make a new dilution and retest using the same strength. The patch test is all right if positive, that is, one or both squares show good firm edema with erythema surrounding it, but a negative patch cannot be relied upon as much as a negative P.P.D. Solutions must be reasonably fresh. The manufacturers advise that P.P.D. always be made up fresh, but we routinely use solutions two to four weeks old. A positive tuberculin test always persists over forty-eight hours, and some men prefer to read at seventy-two and ninety-six hours. If a patient has a negative tuberculin test and has active tuberculosis, the disease is practically always quite far advanced and there is evidence of toxemia, including fever. Most always there are clinical signs

and ample x-ray evidence. There is usually no difficulty in finding tubercle bacilli in the sputum. Therefore, it is safest to call non-reactors non-tuberculous unless acid fast bacilli can be demonstrated.

ACID FAST IN SPUTUM

Acid fast bacilli should be demonstrated in sputum. One of the greatest needs for good tuberculosis work is a good laboratory. There are very few physicians in practice who are competent to do reliable sputum examinations. Many hospital laboratories are also not competent. There are however, some laboratories in practically every state which do good work. Also, in many states the State Board of Health has established either central laboratories or a central, plus branch laboratories, in various sections of the state to which sputum specimens may be sent. Accurate examinations are so important that without reliable information we are hopelessly at sea and we have to abandon 1948 standards and go back twenty years. Sputums should be collected in a sterile jar for a long enough period to secure at least an ounce, if possible. Sputum that is raised early in the morning is the best and the patient must be cautioned to save only that which he feels certain comes from his lungs. Ordinarily, not over a three day sample is desirable for one specimen and for some patients even a three day specimen is not very much. Most laboratories use some sort of concentration method and for this a larger amount is needed than for a simple smear. If only a very slight amount is obtained, a simple smear can be done.

GASTRIC CULTURES

Gastric cultures should be positive for acid fast bacilli and these bacilli must be proven to be pathogenic. Gastric aspirations are easy to do and should be done when there is no sputum or it is scanty. Early in the morning, before breakfast, before food or drink is taken, pass a duodenal tube by the nasal or oral route and aspirate stomach contents. If very little can be gotten add 25 to 50 cc of sterile normal saline and aspirate. The material obtained must be sent to a competent laboratory and examined within a few hours. Good technic for shipping specimens has not yet been standardized and this is a great weakness of the method. Gastric specimens should always be cultured. If acid fast bacilli are grown, animal inoculation should be done as non-pathogenic acid fast bacilli are often found in gastric washings.

THE X-RAY CHEST PICTURE

The x-ray should reveal a parenchymal lesion. The most typical lesions are usually in the upper half of the chest. Very, very seldom do we find positive tuberculosis with no evidence of an x-ray lesion. We have long been in the habit of making a diagnosis of tuberculosis by x-ray alone and even of trying to gauge activity. The x-ray is certainly one of the best methods of diagnosis but we must remember its limitations. After all when we look at an x-ray film we are looking at shadows. For some types of shadows we can be absolutely sure. For another group we can sometimes feel positive and for certain types we can only guess. One of the greatest difficulties that practitioners have is to make good films. It is possible to secure films of very good quality with low powered apparatus but strict attention to technical details must be given. The average film sent in for review is usually over-exposed and under-developed or under-exposed and over-developed; has usually not been washed long enough, often is scratched, marked, or the casette is dirty, or one of many other imperfections can be seen. It is very difficult to make an accurate diagnosis with poor film. Many men seem to think that this is a detail not worth looking into.

PLEURAL EFFUSION

For many years we have regarded idiopathic pleural effusions as definitely tuberculous and there is no reason to change this idea now. If you are a purist you may say that this is tuberculosis of the pleura and not of the lung. Or you will not know whether to call this a minimal or a moderately advanced disease. At first when the effusion obscures the lung it may be impossible to tell whether or not parenchymal disease is present. When the effusion has subsided, further information may be had. However, regardless of these various dis-

tinctions it is always best to call every idiopathic effusion tuberculous and the patient should be treated as such. The new British classification divides tuberculosis into respiratory and non-respiratory. Pleural effusion is in the respiratory group.

To summarize these criteria, a positive diagnosis of tuberculosis may be made if number one, a positive tuberculin is present, plus five, pleural effusion. If number one, positive tuberculin is not present, number two or three, that is, positive sputum on smear or concentrate or positive gastric, must be present, plus four or six. It is rare to have a positive sputum without some x-ray evidence. In sputum examinations, acid fast bacilli are usually reported as rare or many or numerous. Sometimes in a series of ten or twelve sputum samples, one sample will be reported as positive rare. In such cases it is best to have other evidence before making a positive diagnosis. The British disregard the single rare positive. We always do gastrics on such patients and disregard if gastric cultures are negative. We should get more than one positive gastric.

There is one important exception to these criteria. In some hospitals or in some practices, routine tuberculin tests are made at regular intervals followed by x-ray studies. If a patient known to be a non-reactor to tuberculin changes to a reactor (and when change occurs the reaction is usually distinct) and a *persistent* parenchymal lesion appears, a positive diagnosis of tuberculosis is allowable. This patient should have treatment for tuberculosis. Treatment is usually bed rest and there is ample time for further diagnostic procedures.

It is perfectly safe that all patients that meet the criteria given above can be treated for tuberculosis by definative treatment and if possible they should be hospitalized in sanatoria. We are safe in doing pneumothorax, thoracoplasty or using any therapy that seems indicated. Patients fulfilling these criteria should be labeled tuberculous and should be reported to the health department.

TUBERCULOSIS SUSPECT

The next classification is that of *tuberculosis suspect*. This is a difficult classification and this diagnosis should be explained. It includes many that are often now positively diagnosed. When we make a diagnosis of "tuberculosis suspect", we strongly suspect tuberculosis. We cannot make this diagnosis and be complacent about it. In other words, when this diagnosis is made it means that continued study and observation be actively carried on until the diagnosis is clarified. In order to do this it may be necessary to hospitalize the patient for several days or even weeks and in many instances he should be sent to a tuberculosis hospital for special studies. However, when admitted to a hospital for study he should be admitted as a *tuberculosis suspect*. The criteria to be considered in making a diagnosis of tuberculosis suspect are:

1. Tuberculin test is positive.
2. The sputum is negative for tubercle bacilli to smear and concentrate.
3. The culture from gastric lavage is negative.
4. The x-ray shows some parenchymal lesion.
5. Cavity is present.

It is best here to qualify sputum and gastric washing examinations. Our usual routine is to examine three consecutive specimens of sputum. If enough sputum can be collected in a daily sample for concentration, daily specimens may be checked. If this is impossible three three-day specimens are examined. If these are negative or if there is no sputum, three consecutive gastric washings are obtained and cultured. Since it requires approximately four to six weeks before cultures can be read another series of three or even six sputum specimens may be collected and examined.

Tuberculosis suspects should be divided into two main divisions. Group A—those with evidence of toxemia, and Group B—those with no toxemia. As has been said before, the safety of this diagnosis, that is tuberculosis suspect, depends entirely upon the adequacy of the sputum examination. If one is not sure of the laboratory, such patients had better be sent to the sana-

torium or some hospital where adequate studies are available. A patient is placed in this category because study is necessary. He must not be forgotten or neglected. He must be given a very careful complete examination. This examination includes history, chest examination, and other laboratory tests. The presence of fever, pulse acceleration, increased sedimentation rate, rales in the chest, or other abnormal physical findings are evidence of an active pathological process which must be identified. Such evidence of toxemia may or may not be produced by tuberculosis. If one of the many acute infections is responsible, rapid improvement is usually the course. The most common chronic diseases which cause confusion are bronchiectasis, lung cancer, lung abscess, virus pneumonias slow to clear, and various fungus infections. To complete the diagnosis it may be necessary to do bronchograms and bronchoscopic examinations. Serial x-ray studies, seven to ten days apart at first and at wider intervals later are a *must*. Additional sputum studies should be made at least once a month or at six-week intervals. It is usually possible to make a definite diagnosis on these patients with evidence of toxemia in from a month to three months. In that length of time we should be reasonably sure whether the case is tuberculous or not. Patients with a cavity in the upper lung fields are almost always tuberculous and must be studied carefully. It may take some time to get positive sputum, but careful examination will usually disclose bacilli.

The B Group is the group of patients with no toxemia and may present a different picture. Active tuberculosis may be present with little or no evidence of toxemia as measured by clinical findings. The same applies to lung cancer. In this group the same exacting sputum studies and other investigations are in order and serial x-rays are needed. However, in many of these, if after three months of careful observation there has been no change in any of the clinical criteria, if for example, the x-ray shadow has remained the same and there is no evidence of toxemia—the patient may be allowed to go a longer time before another check-up. Intervals may be lengthened to three months or six months. This is the type of case that may be picked up on routine x-ray surveys with an old healed lesion which has long ago been arrested.

There is no known safe rule as to how long patients should go between examinations. If one suspects that tuberculosis might become active, six months is long enough, as a great deal can happen in this period of time. However, all of us familiar with the follow-up of old patients know that there does come a time when infrequent examinations are adequate. When one is convinced that the lesion is inactive, a diagnosis of probable pulmonary tuberculosis, arrested, is safe, but unless at some time a definite diagnosis of pulmonary tuberculosis has been made a diagnosis of pulmonary tuberculosis arrested is not justified.

NON-TUBERCULOUS

The next classification is non-tuberculous. To be sure that a patient is non-tuberculous, first the tuberculin test must be negative. Second, no tubercle bacilli can be demonstrated in the sputum either by sputum concentrate or gastric culture.

Certainly, most patients who have a positive tuberculin test do not and probably never will have active tuberculosis. When a patient does have a positive tuberculin test he should have an x-ray of the chest. History of exposure should be obtained if possible, and if there is any history of exposure within the past two years, he should be re-examined from six months to a year. In clinic practice, a patient with a positive tuberculin test with no exposure to a known case for two years is discharged from the clinic if no x-ray or clinical evidence is obtained. It is known that some of these patients do develop tuberculosis in later years, but the number is insignificant and it is too expensive to follow the entire group. In family practice the physician can keep records of his tuberculin reactors and as a patient returns for various reasons, the chest can be checked as a part of routine studies. Such patients should not be called tuberculosis suspects and they can be given

a clean bill of health for practical purposes, but as their family physician we make a note on their chart not to forget the positive tuberculin.

The patient with a negative tuberculin test and no evidence of toxemia and no physical findings and no x-ray lesions and no acid fast bacilli, can be discharged as definitely non-tuberculous with no medical follow-up needed.

The patient who has a negative tuberculin test, who has a negative sputum, but does have evidence of toxemia or physical findings or x-ray evidence of pulmonary disease should be followed as a general medical problem until the diagnosis can be definitely made. The negative tuberculin test and negative sputum examination fairly well excludes tuberculosis but occasionally follow-up will find that acid fast infection is present. The best examples of this are sarcoid patients who may later develop clinical tuberculosis.

SUMMARY

It is obvious that in the diagnosis of tuberculosis the three principle tools are: (1) The tuberculin test. (2) The x-ray. (3) The sputum examination.

The sputum examination is probably the most important. The tuberculin test is easy to do and x-ray studies can be done fairly easily since equipment is now available in most communities of any size. The great difficulty with x-ray is the poor technic often used. The third part of the study, the sputum examination, *can be done* in remote places but good sputum studies require training, and persistence, and time.

It must be emphasized again and again in tuberculosis work that since we are dealing with an infection we should demonstrate the etiological agent. I do not believe that it is justified to do pneumothorax unless a positive diagnosis can be made. The family physician who has the problem of the follow-up of the survey cases in his own practice, has a great responsibility. If he delays in making the diagnosis all opportunities for good therapy may be lost. He must always remember the possibility of lung cancer being present and that it may masquerade as any other chest condition. Unless the diagnosis is made extremely early there is no chance of successful treatment. He must remember that on survey films many tuberculous lesions are found in minimal stages. If he dilly dallies with the diagnosis and lets the case become moderately advanced or far advanced, there was no use in making the original survey. He should not wait for symptoms to develop before a diagnosis is made. Likewise bronchiectasis should be recognized early. Thousands of children are labeled as chronic bronchitis or sinusitis for years before the true nature of the disease is appreciated.

The chest x-ray is the best screening procedure. Every hospital admission should have this study and it has a high priority in the list of procedures to be carried on in an office practice. With tuberculin tests, routine x-rays, and sputum examinations for every patient who brings up sputum, the family physician will find that he will make many more diagnoses of tuberculosis than he has in the past.

PENICILLIN IN THE TREATMENT OF PEPTIC ULCERS BY CONTINUOUS GASTRIC DRIP*

RICHARD W. YOUNG, M. D.
BATON ROUGE

Since June, 1946 I have been treating peptic ulcers by continuous penicillin gastric drip with a startling degree of success. To date I can report a series of 54 cases treated by this method. Of the 54 cases, 3 were prepyloric ulcers, 51 were duodenal ulcers. No gastric ulcers were treated. This was purposely not done because of the danger of cancer existing in this type of ulcer. Of the 54 cases, 53 gave x-ray evidence and symptomatic evidence of healed ulcer. Three had difficulty in taking the treatment the first time for various reasons. One passed a kidney stone and it was necessary to stop treatment; however, since then, he has successfully taken the treatment. Two

*Presented before the Sixty-eighth Annual Meeting of the Louisiana State Medical Society, Monroe, Louisiana, April 14, 1948.

developed a penicillin rash and it was necessary to stop the treatment. Since then, however, one has been carried successfully through the ten days treatment by using Benadryl. Of the 53 cases discharged as cured none has had a recurrence to my knowledge. The oldest case was discharged from the hospital June 18, 1946.

The method used in these cases was to first establish the existence and exact location of the ulcer by x-ray and fluoroscopic examination. A #12 Levine tube is then passed to the stomach via the nose and fixed in position by tape to the nose. The stomach end of the tube should be at the junction of the lower and middle third of the stomach. The outside end is attached to a 1000 c.c. Abbott drip bottle suspended from an infusion stand. The formula I have used from the first case has not been changed and is as follows: 200,000 units of penicillin dissolved in 1000 c.c. of tap water to which is added 2 drams of soda bicarbonate. This is regulated to drip continuously at the rate of 60 drops per minute which averages 3 bottles in 24 hours. In all cases it is continued night and day for eight, ten or fifteen days. Of the 53 cases successfully treated, 5 were dripped eight days, 45 were dripped ten days, 2 were dripped eleven days and 1 was dripped fifteen days.

In the first few cases nothing was given by mouth at all and daily infusions were given of 5 per cent glucose in 1000 c.c. of normal saline. However, as I found out more about these cases I began to allow the patients to be up during the day as this seemed to eliminate bed weakness. They were then given 10 per cent glucose every other day and allowed to have a glass of sugar water once or twice daily.

The use of vitamins and amino acids was not found to benefit too much. The patients to whom this was given complained of hunger which in turn caused the HCl. content of the stomach to rise and, for this reason, their use was abandoned.

At the end of the treatment x-rays were made again on each case. Of the 53 cases completed, x-ray evidence of healed ulcer was found.

The criteria for time of drip duration were the original size and duration of the ulcer and daily gastric analysis. Samples drawn from the stomach every morning show a rapid fall in the total and free HCl. the first three days to no HCl. and 4 to 12 total acidity and remaining so throughout treatment unless some disturbance occurs. In one case I noted a rise of HCl. on the fifth day and sixth day. I questioned the patient carefully and found he had been rinsing out his mouth with coffee. In another case a rise in HCl. was traced to chewing gum. All cases are taken off tobacco during the treatment. The obvious reason, of course, is to facilitate acid reduction. It is a well known fact that penicillin is partly destroyed by HCl.

An interesting observation was made early in this series of cases, and may be one of the important factors in its success. That is the cessation and abscence of active peristalsis. It was noted at the end of ten days that barium from the first x-ray taken the day the treatment was begun was still in the colon with no apparent discomfort. All the cases having pain usually subsided entirely by the second or third day. In those cases with an extremely high acid content more than 2 drams of soda bicarbonate may be added at the beginning of the treatment.

The adjustment of the Levine tube is very important as the days pass by because the empty stomach will shrink and draw up. The tube, of course, will not shrink and is apt to slip out into the duodenum. This can be detected by daily sample taking from the tube or by fluoroscopy. Should bile appear in the sample, the tube should be moved up until it is again in the stomach at the junction of middle and lower third of the stomach. This step is very important; healing will not occur if the end of the tube is past the ulcer. This occurred in one case and the patient had to be dripped over under more careful observation, with a successful healing.

In conclusion, I would like to emphasize

that this form of application of penicillin has only been used on 54 cases and I, therefore, can only give it to you for what it may be worth. I do feel, however, that the degree of success obtained in this series is worthy of further study and use. The simplicity of the treatment and the lowering of the morbidity rate are the important factors in its favor. To date none of the cases reported has had a recurrence. No controls were run. Since the cases were seen in private practice, this was impractical. However, there was no mortality, a point worthy of recognition.

REFERENCE

Young, Richard W.: New method of treating peptic ulcers with penicillin gastric drip. Preliminary report. New Orleans M. & S. J., 100:379, February 1948.

SOME DATA PERTINENT TO THE DIAGNOSIS AND PROGNOSIS OF PULMONARY TUBERCULOSIS

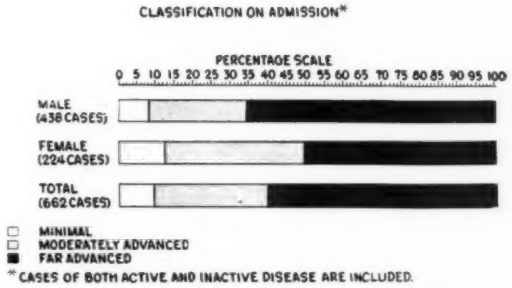
PHILIP B. JOHNSON, M. D.†
AND
LOUIS A. MONTE, M. D.†
NEW ORLEANS

During a five year period ending in 1944, 662 patients with tuberculosis of the lungs were admitted to our unit of the Dibert Memorial (white) tuberculosis service of the Charity Hospital. In the great majority of the cases the disease had been recognized or suspected only a short time before the patients were sent to the hospital, or its existence had not been suspected prior to their admission. The patients were referred by physicians from all parts of the state of Louisiana. It is probable, therefore, that the relative numbers of cases of minimal, moderately advanced, and far advanced disease in this group of patients reflects with a fair degree of accuracy the stages at which tuberculosis was usually recognized throughout the state during this period.

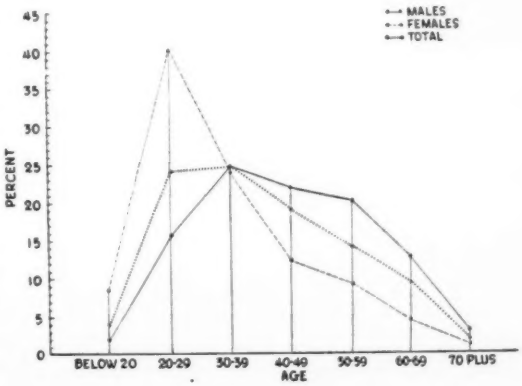
Further, analysis of the clinical pictures presented by these patients and study of the cirmumstances which led to the diagnosis should yield information which might be of value in the diagnosis of future cases, and which might point the way toward recognition of the disease at an earlier stage. Finally, observation of the courses of these patients after their admission and determination of their fates have shown us what we were able to accomplish in the treatment of tuberculosis in various stages of advancement.

STAGE OF THE DISEASE

The extent of the pulmonary lesions (Figure 1) was minimal in approximately


CLASSIFICATION ON ADMISSION*
PERCENTAGE SCALE
0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100
MALE (438 CASES)
FEMALE (224 CASES)
TOTAL (662 CASES)
MINIMAL
MODERATELY ADVANCED
FAR ADVANCED
* CASES OF BOTH ACTIVE AND INACTIVE DISEASE ARE INCLUDED.

10 per cent of the cases, moderately advanced in 30 per cent, and far advanced in 60 per cent. On the average, the disease was recognized at an earlier stage in women than in men, there being slight but probably significant differences in the incidence of each of the three stages. It is also of interest that on the average the disease had become apparent at a distinctly earlier age in women (Figure 2). It should



*Presented before the Sixty-eighth Annual Meeting of the Louisiana State Medical Society, Monroe, April 14, 1948.

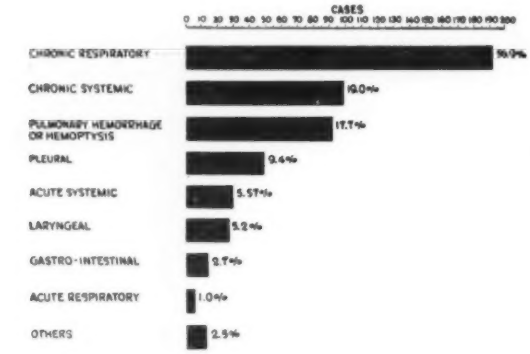
†From the Department of Medicine of the Louisiana State University School of Medicine and the Charity Hospital of Louisiana at New Orleans.

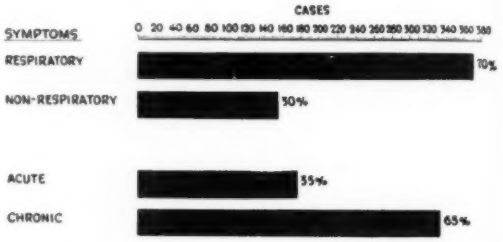
be noted that the curves for males and females cross in the fourth decade. Three-fourths of the women but less than half of the men were under the age of forty; 10 per cent of the females but only 2 per cent of the males were less than twenty years of age. In the entire group, half of the patients were older than forty years of age, and 13 per cent were aged sixty or above.

DIAGNOSTIC DATA

Adequate information regarding the symptoms or circumstances which led to the diagnosis is available in 583 of the cases. In 509 of these, tuberculosis was suspected on the basis of the symptoms of which the patients complained, but in 74 the disease was picked up "accidentally," this term here being applied to the detection of pulmonary tuberculosis by x-ray examinations of persons in whom there were no symptoms or signs which aroused the suspicion of the existence of this disease.

The most prominent complaints or types of symptoms in the symptomatic cases are depicted in Figure 3, in the order of the frequency of their occurrence. Here it is noted that presenting symptoms were chronic respiratory in slightly over one-third of the cases, chronic systemic in one-fifth, onset with hemoptysis in about the same number, pleural in approximately one-tenth, acute systemic manifestations in approximately 6 per cent, hoarseness in about the same number, gastrointestinal in about 3 per cent, and acute respiratory in 1 per cent. It should be noted further that the principal complaints in almost a third of the cases were not suggestive of disease of the respiratory system, and that in about the same number the presenting symptoms were acute rather than insidious in onset. (Figure 4).

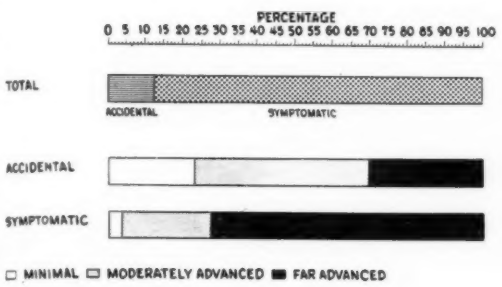




An attempt to relate the type of presenting symptoms to the stage of the disease revealed no definite correlation between the two, but it should be stated that in those patients who had symptoms due to tuberculosis the disease was at a minimal stage in only 4 per cent. Although no detailed study of the physical signs was made, it is relevant to state that examination of the lungs revealed only normal findings in nearly all patients with minimal lesions and in many of those with more extensive disease.

It should be of interest to record how the x-rays came to be made in the cases which were discovered "accidentally." Twenty-three cases were picked up during the course of studies for supposedly non-tuberculous disease, 23 during examinations for induction into military service, and 17 by x-raying persons known to have had intimate exposure to tuberculous individuals (contact studies); the other 11 cases were detected by x-rays taken prior to employment, following trauma to the chest or shoulder region, or for other miscellaneous reasons. In 1 patient who had undergone an appendectomy the discovery of tuberculous lesions of this organ led to the x-ray study, which demonstrated the presence of active pulmonary tuberculosis.

Figure 5 shows the percentage of cases picked up "accidentally" as contrasted with the symptomatic cases. Roughly 7 out of 8 cases were recognized on the basis of symp-

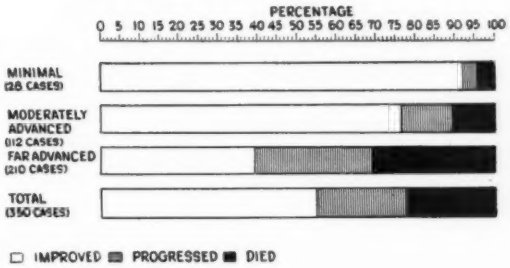

PERCENTAGE
0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100
TOTAL
ACCIDENTAL
SYMPTOMATIC
ACCIDENTAL
SYMPTOMATIC
MINIMAL MODERATELY ADVANCED FAR ADVANCED

toms. The two lower blocks show the incidence of minimal, moderately advanced and far advanced disease in "accidental" (middle block) and symptomatic cases (lower block). It is seen that the incidence of minimal disease was much higher in the "accidental" than in the symptomatic cases, and the reverse obtains for far advanced disease in the symptomatic cases. The 17 cases of minimal disease picked up by accident or by routine x-rays comprise almost half of the total number of cases of minimal tuberculosis.

PROGNOSTIC DATA

The patients in this series were observed for periods varying from a few days to more than five years. During the periods of observation 176 patients died, giving a gross mortality of 30 per cent in the cases of active tuberculosis. The mortality in cases of minimal, moderately advanced, and far advanced disease was respectively 2½, 12, and 40 per cent.

Two hundred and thirty-three of the patients died shortly after admission or did not remain for treatment under our care for various reasons, leaving a total of 350 patients with active tuberculosis who lived more than three months and whose courses under therapy we were able to follow. Of these, the disease improved or became arrested in slightly over one-half, failed to improve or progressed in one-fourth, and was fatal to a fourth. Figure 6, which shows the results of therapy in these patients, reveals that the percentage of cases in which improvement or arrest occurred decreases from 90 to 75 to 40 as the stage of the disease advances, and that the mortality increases from 4 to 11 to 30 per cent. Nearly all of the patients with minimal disease did well. On the whole, those with more extensive disease improved only when it was possible to effect collapse of the lesions by pneumothorax or thoracoplasty, but were able to accomplish this in only two-thirds of the patients with moderately advanced disease and about a third of those with far advanced lesions.


PERCENTAGE
0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100
MINIMAL (28 CASES)
MODERATELY ADVANCED (112 CASES)
FAR ADVANCED (210 CASES)
TOTAL (350 CASES)
IMPROVED PROGRESSED DIED

On the basis of previous experience, we estimate that among this group of patients the eventual mortality due to tuberculosis will be in the neighborhood of 50 per cent. We anticipate an ultimate mortality rate of about 10 per cent in the cases of minimal disease, 40 to 50 per cent in the moderately advanced, and about 80 per cent in the far advanced cases. The corresponding rates of eventual cure of tuberculosis we expect to be in the neighborhood of 90, 40 and 15 per cent. Cure in most of the cases of minimal disease will be effected by relatively short periods of rest, supplemented by pneumothorax of short duration in some cases. On the other hand, those with more extensive lesions who are fortunate enough to recover will reach the stage of apparent cure only after prolonged periods of absence from their homes and their work, and in most cases after prolonged or permanent collapse procedures have been employed.

DISCUSSION

If the results of this survey are applied to conditions which existed throughout the state during the period which it covers, it may be said that in most cases of tuberculosis the disease had progressed beyond a stage at which the chances of recovery were good before the disease was recognized. The cause of this delay in diagnosis is obvious:

It was not because physicians failed to recognize the clinical picture of tuberculosis, but rather because in most patients the disease had progressed beyond the minimal stage before significant symptoms appeared. This is apparent from the fact that although the first physician seen promptly made the diagnosis in the great majority of the symptomatic cases, the disease was already moderately or far advanced in 96 per cent of these cases. It appears that on the average, women may have sought medical advice earlier than men, which may explain the discovery of the disease at less advanced stages in females. The relatively large numbers of elderly persons is probably related to the small number of cases of minimal disease, or more specifically to the fact that many of the patients had had tuberculosis for a long time.

The obvious corollary of these results is the fact that tuberculosis in its early stages is usually not symptomatic, and that the disease can be detected while involvement is yet minimal only by x-ray studies of presumably healthy persons and of patients with symptoms which seem to be due to other conditions. The findings emphasize that acute as well as chronic illness may be due to tuberculosis, and that in many cases the presenting symptoms are not suggestive of trouble with the lungs.

It should be noted that there has been a slight but significant increase in the percentage of minimal, favorable cases admitted to our service during the past several years. A decade ago less than 2 per cent of the patients had minimal disease;[1] during the period of the present survey this had increased to 10 per cent. It is certain that the present programs of the State Board of Health and the Louisiana Tuberculosis Association will result in continued betterment of the situation, but in our opinion these programs can fulfill their purpose only if the practicing physician cooperates with them fully, and in addition carries on his own case-finding program among the persons who have chosen him as their medical advisor. If the doctor says to get an x-ray, it is made, but many persons are not influenced by placards or other impersonal means of exhortation.

SUMMARY

Of the white patients with pulmonary tuberculosis admitted to our service during the period 1940-1944, approximately 30 per cent have already died, and we anticipate that ultimately at least half of them will die of tuberculosis. Only about 40 per cent will eventually reach the status of apparent cure. These poor results are in large part due to delay in the diagnosis of this disease, so that in the majority the lesions had progressed beyond stages readily amenable to treatment, before the disease was recognized. The cause of this delay lay not in the inability of physicians to recognize tuberculosis when it gave rise to symptoms, but to the fact that in its early stages it often produced no symptoms. The hope of increasing the recognition of tuberculosis in its early stages therefore lies not so much in greater suspicion of this disease in patients whose symptoms are compatible with its presence, but rather in establishing the presence or absence of tuberculosis (and other diseases of the chest) by x-raying presumably healthy persons in increasing numbers at periodic intervals. The immediate hope of greatly increasing the number of cures lies not in any momentous advances in the methods of therapy, but rather in the recognition of more early, incipient cases. There is already a cure for minimal tuberculosis; there is no cure in sight for most of the cases of far advanced disease.

REFERENCE

1. Monte, L. A. and Blitz, O.: Factors delaying the diagnosis of pulmonary tuberculosis. New Orleans M. & S. J. 90: 468, 1938.

DERMATOMYOSITIS*

REPORT OF A CASE

W. H. BROWNING, M. D.
SHREVEPORT

Dermatomyositis is not a rare disease. Several hundred cases have been reported. Yet, it is sufficiently infrequent and its manifestations are so variable, especially in

*Read before the Sixty-eighth Annual Meeting of the Louisiana State Medical Society, Monroe, April 14, 1948.

the early stages, that a diagnosis may be easily overlooked.

The cause of dermatomyositis is not known. Several theories concerning the etiology have been advanced, but none has been substantiated. Banks[1] has emphasized that dermatomyositis, scleroderma, disseminated lupus erythematosus, Libman-Sacks syndrome and periarteritis nodosa, though unrelated in clinical features, have a close similarity in detailed pathology. He suggests that these conditions require evaluation for a possible relationship or a common denominator among them. McCombs and MacMahon[2] and Lowell[3] in recent articles, have suggested that an allergic mechanism may be responsible for these entities. McCombs and MacMahon[2] point out that asthma and eosinophilia may occur in association with dermatomyositis. It has been suspected for some time that periarteritis nodosa is the result of or is related to the hypersensitive state.

The clinical and pathologic manifestations of dermatomyositis have been fully described in recent literature[1, 2, 4, 5, 6, 7, 8, 9] The following case report will serve to demonstrate most of these manifestations.

CASE REPORT

Mrs. E. B. S., housekeeper, 66 years of age, came to my office on July 18, 1946.

Chief Complaint: Rash on face and forehead.

Present Illness: About June 1, 1946, patient developed a rash on her forehead. It was rather red, scaly, and itched. About a week later it spread to her face. Her face and eyes had been swollen on several occasions, and she had had severe urticaria twice during this interval. One week prior to my examination she had consulted a local dermatologist and had been given x-ray treatment.

Patient had been troubled with "acid stomach" for several years and on several occasions had consulted a local internist who never found any organic trouble but treated her with alkalies. At the onset of the present illness her digestive symptoms became worse. She described the symptoms as "something boiling in the abdomen".

During this illness she had experienced what she called heart attacks. The last one was two days before she consulted me. She had the sensation of the heart turning over and then stopping. Following this, she would become weak and break out in a profuse sweat.

For many years she had had "sinus trouble". She sneezed excessively upon retiring and upon arising. The sneezing was in paroxysms. She coughed a great deal especially upon going to bed and upon arising. She coughed quite a bit during the night and had some wheezing and rattling in the chest. She had a troublesome postnasal drip and one nostril or the other was blocked most of the time. She had never had a severe case of asthma, but did have to sit up in bed at times because of coughing.

She had severe constipation and had been taking Ex-Lax for several years. Often she had frontal headaches which were not severe, but annoying.

Past History: She had severe urticaria as a child, but seemed to outgrow this except that she had an occasional urticarial wheal from time to time. After the onset of the present illness she had two severe attacks of urticaria. She had an appendectomy in 1921 and a tonsillectomy in 1931. She had not had any serious illnesses.

Menstrual History: Menopause at the age of 40. She was nervous and upset for about 10 years. She was the mother of several children.

Family History: Negative for allergy, cancer, tuberculosis, and heart disease.

Physical examination:

Temperature: 100.6° F. (Patient did not realize that she had fever.)

Blood Pressure: 170, systolic; 90, diastolic.

Weight: 126 pounds.

Skin: She had an erythematous, macular rash on the forehead, face, neck, chin, left ear, and scalp. These areas were covered with fine, light colored scales. The involved skin seemed to be somewhat endurated.

Mouth: Teeth false, Mucous membrane normal.

Throat: Tonsils removed. Mucous membrane normal.

Nose: Mucous membrane swollen and red. Almost complete obstruction on right side.

Eyes: Pupils reacted to light and accommodation. The fundi appeared to be normal.

Ears: Used hearing aid. Drum membranes retracted. Left auditory canal red and scaly.

Sinuses: Right antrum slightly hazy.

Neck: No abnormal pulsations.

Heart: Approximately normal in size. Rhythm was disturbed by numerous premature contractions. Sounds were clear and distinct.

Lungs: Percussion note normal. Expansion free and symmetrical. Numerous coarse rales at the base of both lungs. Breath sounds normal.

Abdomen: Relaxed. Midline surgical scar. Generalized tenderness. No rigidity. Spleen palpable, smooth, and fairly firm. Liver palpable. Some ptosis of right kidney, but no tenderness over either kidney.

Glands: Thyroid not enlarged. Anterior cervicals, posterior cervicals, epitrochlears, inguinals, and axillary not enlarged.

Reflexes: Biceps and patella normal.

Laboratory Studies: July 18, 1946, Red cell

count 4,830,000; white cell count 6,600; hemoglobin 13 Gm.; polymorphonuclear leucocytes 50%; small lymphocytes 40%; eosinophiles 10%. Malaria plasmodia were present. Skin test and agglutination test for undulant fever negative. Kahn test negative. Fractional gastric analysis well within normal limits. Urine: Acid reaction, specific gravity 1.010, albumin 1 plus, pus cells 0 to 15/hpf. Sugar, bile, acetone, red cells, and casts negative. Another specimen of urine was examined on July 19, 1946, and it showed a trace of albumin and pus cells 2 to 5/hpf.

Impression:

1. Dermatitis from ingestion of phenolphthalein (Ex-Lax).
2. Malaria.
3. Urticaria, cause to be determined.
4. Perennial hayfever.
5. Allergic bronchitis.
6. Probable gastrointestinal food allergy.

An allergic survey was done. She did not react to pollens. Of the common inhalants she reacted to house dust (Endo) and wool. She was patch-tested with soaps, cosmetics, and other common contactants. Reactions were conspicuous by their absence. Food tests showed reactions to chicken, corn, lima beans, black-eyed peas, sweet potato, red pepper, grapefruit, tuna fish, and peaches. She knew that peaches and grapefruit caused urticarial wheals and gas and burning in the abdomen.

Progress: She was given quinine sulphate on July 22, 1946, 10 grains, night and morning. She became nauseated and vomited. After two days the dosage was reduced to 10 grains at night. She continued to vomit. The quinine was discontinued on July 29, 1946, and improvement was excellent. She did not have fever and the skin condition was clearing satisfactorily, but on August 7 she began to run fever again and was given Atabrine. The fever stopped and her skin cleared up except for a slight redness. She felt better.

She lived out of town, but reports by a daughter who lived in Shreveport indicated that she was doing well. On October 1, patient wrote that she had some "breaking out" on arms and feet, and that the feet were swollen. I advised that she come in for a check-up, but this was not done until October 29. At this time the arms and feet were clear. The skin of the face and forehead showed slight redness. She complained that her hair was falling out. The scalp appeared to be normal except for a few fine scales. Temperature was 98°F., blood pressure 150/80, weight 126 pounds. Liver and spleen were palpable. Urine: specific gravity 1.018, acid reaction, and otherwise normal. The red blood cell count was 3,210,000. Hemoglobin 7.8 Gm. White count 9,650. Polys 61%. Small lymphocytes 32%. Eosinophiles 7%. She was started on ferrous sulphate 0.25 Gm. t.i.d. She was, also, given suggestions about her diet.

A letter on November 5 stated that she was having fever ranging from 100° F. to 102° F. She complained of weakness and stated that the loss of hair was pronounced. She complained of numbness in the fingers. They turned pale and after the color began to come back it felt as though the bones would burst. During the morning of this date her entire bory had turned extremely red and remained this way for thirty minutes. Other than this, the skin condition was very satisfactory.

About November 15, she began to swell. It was very bad in feet and legs, but by November 21 when patient came to my office she had edema of face, upper extremities, trunk, and lower extremities. She was not having fever. Urine: Specific gravity 1.018, albumin 2 plus, many pus cells; otherwise negative. Blood: Red cell count 4,160,000; hemoglobin 9.2 Gm.; white count 9,950. Polys 60%. Small lymphocytes 30%. Eosinophiles 10%. Total blood protein 8.8 mg. Albumin 2.9 mg. Globulin 5.9 mg. She was hospitalized and given Amigen intravenously daily for several days and was kept on a high protein diet. It was difficult to get her to eat meats. She was started on a vegetable diet in 1930 or 1931 because of "acid indigestion." To a large extent she had been on this diet ever since and had lost her desire for meats.

On December 4, after patient had slept under a wool blanket, the skin flared up. The rash was on face, neck, arms, forearms, hands, legs, feet and to a lesser extent on the thighs and abdomen. It was very red. The edema had completely disappeared. December 5: Total protein was 9.3 mg. Albumin 3.7 mg. Globulin 5.6 mg. She continued to improve and gained considerable strength. On December 12, the rash had completely disappeared from her body. She had a little on her cheeks which was dry and scaly. Total protein 9 mg. Albumin 5.6 mg. Globulin 3.4 mg. She returned to her home.

On January 4, 1947, patient wrote that she had no rash except a little on the neck and face. She, also, reported that she had no swelling, but that she had had severe asthma the preceding night. She had to sit up most of the night, but had not taken epinephrine. Until this time cough and nasal symptoms had been very much improved.

January 8, 1947, patient wrote that she was having fever, the feet and legs were breaking out, she ached all over, had chilly sensations and felt very weak. She asked, "Is it possible that a blanket, with a small amount of wool, on the bed could be causing this?" I had previously advised her to avoid wool. In reply I again advised her to avoid wool.

January 16, 1947. Temperature: 98° F. Blood pressure 168/80. Weight 120 pounds. Spleen palpable. Ankles slightly edematous. Skin of face red. Eczematoid patches on legs. Skin was

otherwise clear. Urine: Albumin 2 plus, a few hyaline casts. Red cell count 3,620,000. Hemoglobin 9.6 Gm. White cell count 13,500. Polys 60%. Small lymphocytes 31%. Eosinophiles 9%. Total protein 9.6 mg. Albumin 4 mg. Globulin 5.6 mg.

January 23 she wrote that she was having fever, less than 100°, coming on about 3 P.M. She stated, "I am gaining strength fast."

February 1 she wrote that she had sores on her legs which were very painful. The preceding night she had urticaria on left hip.

February 10 she had quite a bit of rash on scalp and legs, and in places there were rather large scab formations. The underlying area was ulcerated. She had lost most of her hair. Temperature 98° F. Blood pressure, 150/80. Weight 124 pounds. Urine: Specific gravity 1.022. Albumin 3 plus. Otherwise negative. The blood count was very similar to previous one. Total protein 9.8 mg. Albumin 4.5 mg. Globulin 5.3 mg. She had no edema. She was advised to use hydrogen peroxide and boric acid for removing scales, and after removal of scabs to apply Tyroderm.

On February 15, she wrote that her hands and arms were breaking out, "with a red, red rash, that itches something terrible."

On March 12 she wrote that she had gained 12 pounds, did not have any swelling and that her skin was clear except for some dark spots on her legs.

On April 7 she wrote that she was having rheumatism or arthritis. She stated that it had started about five weeks previously in the fingers. They became very blue and numb. The blueness and numbness would come and go, but was getting worse. The fingers were painful and the pain radiated to the shoulders. She stated that she was having pains in both knees, pain in the muscles or nerves, and she emphasized that the pain was severe. She stated that her skin was about well except she had scars on arms and legs, however, she stated that they still itched. She, also, stated that she was "still breaking out with those large whelps."

April 15 patient came to the office. The wrists, knees, shoulder joints, and right ankle were very swollen and tender. She also had swelling of the proximal interphalangeal joints and the metacarpophalangeal joints. The swelling was typical of rheumatoid arthritis. The fingers were very blue. The scalp was covered with a new growth of hair. The skin on the outer surfaces of the legs had many areas of hyperpigmentation. It was in splotches of various sizes, of a brownish color, and at first glance, had the appearance of bronzing, but on closer examination appeared too dull. The skin between the areas of hyperpigmentation had a glazed, atrophic appearance. The spleen was palpable. Temperature 98° F. Blood pressure 170/90. Weight 125 pounds. There was no edema except at the joints. Urine: Specific gravity 1.020. Albumin 3 plus. Occasional pus cell, otherwise negative. Blood: Red count 4,580,000. Hemoglobin 10.4 Gm. White count 9,400. Polys 80%. Small lymphocytes 13%. Eosinophiles 7%. Blood calcium 9.3 mg./100 cc. Phosphorus 5 mg. Total protein 7 mg. Albumin 2 mg. Globulin 5 mg. NPN 40 mg. She was given Amigen intravenously and seemed to improve. Most of the swelling left the joints, and they became less painful. X-ray examination of the skull and wrist joints was done April 22, 1947. The report of Dr. C. P. Rutledge, radiologist, was: "X-ray examination of the skull shows no evidence of fracture or other gross bone changes. The sella is of the semiclosed type and above the average in size. The clinoid processes show evidence of fragmentation suggestive of intrasella pressure. Wrist: Some demineralization is noted in both wrist joints, with narrowing of the joint spaces, which is more or less characteristic of an atrophic arthritis." No further studies were done relative to the fragmentation of the clinoid processes.

On May 7 biopsy was taken over and from the right deltoid muscle. The report of Dr. W. R. Mathews, pathologist, was:

"**Gross:** There are three masses of tissue. One is a fusiform excision of skin and subcutaneous tissue that measures as much as 1.4 cm. in width, 2 cm. in length, and 2 cm. in thickness. The skin is free from tumor, shows so far as I can tell normal color and is somewhat atrophic in appearance. The other two masses appear to be from muscle and measure 1 x 1 x 1½ and 1 x 1½ cm.

Microscopic: Dermatomyositis

1. Sections from the skin show atrophy and hyperkeritinization of the epidermis, proliferation, fibrinoid degeneration and perhaps slight edema of the collagenous tissue of the corium, thickening of the walls of the small vessels and perivascular infiltration with lymphocytes, plasma cells and monocytes. Also, one notes similar changes in the collagen and small vessels of the subcutaneous fat.

2. The muscle fibers are swollen and hyalinized, show no cross striation and there is proliferation of the sarcolemmal sheaths. The interstitial tissue is infiltrated with lymphocytes, plasma cells, and monocytes, especially around vessels and there is moderate fibroblastic reaction but no dense fibrosis as yet. The walls of the small arteries are thickened and in some instances show intimal hyalinization."

Patient returned to her home on May 13. She seemed to improve until July 1 when she developed a severe cold. On July 2 she had massive consolidation at the base of the right lung and some consolidation at the base of the left lung.

She expired on July 12, 1947, as a result of pneumonia.

DISCUSSION

Dermatomyositis is a disease of unknown etiology, but it has been suggested that an allergic mechanism may be the underlying cause. A typical case has been presented showing most of the various manifestations, and it occurred in a patient who had allergic symptoms all of her life and continued to have symptoms throughout the course of this disease. Dermatomyositis has a mortality rate of about 50 per cent and death is usually due to an intercurrent infection. Pneumonia was the cause of death in the case presented.

The case presented by McCombs and MacMahon[2] was suspected of having a drug rash. Other cases have been suspected of being contact dermatitis. In the case presented it was first thought that a drug was responsible for the rash. Later it was thought that wool might be the cause.

Diagnosis probably could have been established sooner had the true nature of the disease been thought of. However, biopsy does not always insure a positive diagnosis. Healthy muscle may be contiguous with diseased muscle, and if healthy muscle is obtained at biopsy the diagnosis would be extremely difficult.

The development of nephritis, rheumatoid arthritis, and Raynaud's phenomena during the course of the illness indicates that the systemic reaction is severe. The vascular lesions of this disease are a prominent feature, and if allergy is responsible for these changes, present methods of diagnosis are inadequate. Research work dealing with tissue reactions in the human subject as well as cultured tissues is strongly indicated.

REFERENCES

1. Banks, Benjamin M.: Is there a common denominator in scleroderma, dermatomyositis, disseminated lupus erythematosus, Libman-Sacks syndrome, and polyarteritis modosa? New England J. Med., 225:443, 1941.

2. McCombs, Robert P. and MacMahon, Edward H.: Dermatomyositis associated with metastasizing bronchogenic carcinoma. A Clinicopathological Conference. Med. Clin. North America, 1148, Sept., 1947. (Boston)

3. Lowell, Francis C.: The newer concept of allergy to drugs and bacteria, J. A. M. A., 136:665, 1948.

4. Marcus, I. H. and Weinstein, J.: Dermatomyositis: Report of case with review of literature. Ann. Int Med., 9:406, 1935.

5. O'Leary, P. A. and Waisman, M.: Dermatomyositis: Study of forty cases. Arch. Dermat. & Syph., 41:1001, 1940.

6. Keil, H.: Manifestations in skin and mucous membranes in dermatomyositis, with special reference to the differential diagnosis from systemic lupus erythematosus. Ann. Int. Med., 16.828, 1942.

7. Freudenthal, W.: Generalized scleroderma and dermatomyositis: Histologic comparison. Brit. J. Dermat., 52:289, 1940.

8. Hecht, Manes S.: Dermatomyositis in childhood. J. Pediat., 17:791, 1940.

9. Silverman, Jacob J. and Powell, Vernon E.: Peripheral vascular changes in dermatomyositis, Am. Heart J., 30:441, 1945.

DISCUSSION

Dr. J. D. Youman, Jr. (Shreveport): Dr. Browning is to be congratulated on his presentation of this case of dermatomyositis, associated with respiratory allergy.

Dermatomyositis is an acute, subacute, or chronic disease of unknown origin, characterized by a gradual onset with vague and indefinite prodromes, followed by edema, dermatitis and multiple muscle inflammation.

The dermatitis may resemble erythema, erysipelas, urticaria, eczema, or erythema nodosum, as well as disseminated lupus erythematosis and early scleroderma. The face, especially the eyelids and the extremities, particularly the proximal portions and over involved joints are involved. Fever is moderate and remittent or intermittent. Enlargement of the spleen is frequently seen. Creatinuria is present, due to structural damage in the muscles.

Treatment is symptomatic. Some of the patients undergo spontaneous cure and others progress, as did Dr. Browning's, to final death from intercurrent infection.

The etiology of dermatomyositis is unknown and Dr. Browning suggests that an allergic mechanism may be the underlying cause. If by that, he means allergy to infection, as we feel rheumatic fever is, then I will agree with him. Several case reports that I have seen, tend to favor this idea, or that of an actual infection, itself. Omens, in 1944, reports the disease in a boy thirteen years old, who, because of progressive weakness, was even unable to raise his head from the bed. Sulfonamides caused no appreciable results. Penicillin was administered totalling 1,200,000 units; 200,000 units the first day by vein and thereafter 5,000 units every four hours by intramuscular injection, a very inadequate dosage, as we know today. Fifteen months later, when this case was reported by Omens the boy was able to get about with the aid of support. The exanthem had faded somewhat, but the edema was still present.

Madden, in 1944, reports an early case manifesting extreme laryngeal and pharyngeal edema, dermatomyositis of the extremities, and dermatitis of the face. He was given penicillin, 100,000 units daily. An improvement in his voice and

physical status was seen at the end of the third day. There was a decrease in the laryngeal edema. At the end of ten days, the cutaneous changes were almost gone. At the end of three weeks, there was no edema and the changes in the eyelids had entirely disappeared. He commented on this case, "This boy has had remarkable results with penicillin. The symptoms reappear in about forty-eight hours when penicillin is stopped." This sounds like what occurs in subacute bacterial endocarditis. The penicillin just does not have the power to completely eradicate the infection.

Andrews also reports a case in which penicillin was of help.

Benson Cannon, in 1944, reported a case with remarkable improvement after large doses of vitamin C, vitamin E; thyroid therapy, cod liver oil, and x-ray therapy.

Maurice Costello believes that dihydrotachysterol is of great value in these cases.

Both of these cases present the endocrine angle, which some believe to be the cause.

Dostrovsky and Sogher reported two cases, both of whom had malignant tumors. They concluded that there is a relationship between dermatomyositis and malignant tumors. The appearance of the dermatomuscular symptoms shortly after the development of the tumor, and moreover, their intensification on metastasizing of the tumor, thereafter persisting until the patients succumbed in a state of carcinomatous prostration, would uphold their views.

Herbert Rattner reported a case associated with carcinoma. In Dr. Browning's case, x-ray of the skull showed an unexplained fragmentation of the clinoid process, suggestive of intrasella pressure.

The total white counts in these cases is usually within normal limits, as was so with Dr. Browning's case. They do not, however, show an eosiniphilia, as is present in the case presented. I believe this was due to the associated respiratory allergy and not related to the dermatomyositis.

We find ourselves again back where we started. We do not know the cause of dermatomyositis, but it is through such reports as Dr. Browning's, suggesting allergy, others suggesting an endocrine basis, those suggesting infection, and those suggesting malignancy that finally the answer will be unraveled.

SYMPATHETIC OPHTHALMIA

REPORT OF A CASE

WILLIAM W. HART, B. S., M. D.
SHREVEPORT

Case presented is one of sympathetic ophthalmia which developed as a complication of a perforating wound of the right eye.

REPORT OF CASE

A 48 year old negro female was admitted to the Charity Hospital in New Orleans, Louisiana, on August 2, 1946, complaining of pain in both eyes and impairment of vision in both eyes. She gave a history of receiving a perforating wound in the right eye on June 3, 1946, when a bottle of beer exploded and a piece of glass hit her right eye and perforated the cornea.

She was treated by her local physician who closed the perforating wound of the right eye with corneal sutures and a conjunctival flap. The wound healed, but the right eye continued to pain and remained injected and sensitive to light with marked lacrimation all the time. On July 30, 1946 she developed redness and pain in the left eye and noticed some impairment of vision in left eye.

On admission to the Charity Hospital temperature was 98.6° F., respiration was 16, blood pressure was 110/60. General physical examination was negative.

Examination of right eye: Vision right eye, light perception. There was moderate injection, both deep and superficial of the conjunctiva. There was a healed laceration of the cornea extending from the limbus across the cornea from 9 o'clock to 3 o'clock. There was an incarceration of the iris in the corneal laceration. Lens capsule was ruptured and the lens was opaque.

Slit lamp examination right eye: Numerous keratic precipitates, definite aqueous flare with cells in the anterior chamber.

Funduscopic examination right eye: Unable to see fundus due to lens opacity.

Examination left eye: Vision 20/80. There was moderate edema of lids and conjunctiva. Moderate injection of the palpebral and bulbral conjunctiva. Cornea was clear. Pupil was irregular with many posterior synechia. Media was clear grossly.

Slit lamp examination left eye: Aqueous flare. Posterior synechia present with some uveal pigment deposit on anterior lens capsule.

Funduscopic examination left eye: Vitreous slightly cloudy. Disc margins were indistinct with definite elevation. Mild AV notching. There was definite stippling in

the macular area with some disarrangement of pigment.

Tentative diagnosis: Sympathetic ophthalmia OS.

Laboratory Report: RBC 4,500,000; Hemoglobin 14 grams; WBC 5,240; Differential: 58% Polymorphonuclears, 40% Lymphocytes, 2% Monocytes. Blood Wasserman negative; Urinalysis negative; Blood Chemistry: urea 14 mg.; glucose 114 mg.; X-ray of chest negative; EKG negative; X-ray of sinuses negative. Dental check up was made and dentist recommended extraction of all teeth, which was performed.

Treatment: Treatment consisted of: Atropine 1% gtts. 1 OU t.i.d., Penicillin, units 25,000 q 3 hours for 5 days. Patient was given 100 grains of sodium salicylate and calcium gluconate 7½ grains daily. She was also given 5 milligrams of Vitamin B_1 t.i.d.

On August 4, 1946 enucleation of OD was performed under local retrobulbar anesthesia.

Patient was started on intravenous typhoid, first dose, 30,000,000 killed typhoid bacilli given intravenously and was increased 15,000,000 every other day for eight doses. There was a moderate response to this with temperature rising to 101° F. after each injection. It was then deemed advisable to give this patient artificial fever therapy. Artificial fever therapy was started September 3, 1946 and she received ten treatments. Temperature range on the fever therapy was 103 to 105° F. for two to three hours each time. The interval between treatments was five days.

Pathological Report—Gross: The specimen consists of a firm eye measuring 25 x 24.5 x 24.5 mm. An opaque band scar extends across the cornea from 2 to 9 o'clock. There are folds of the retina and thickening of the choroid posteriorly. Engorged blood vessels radiate from the nerve head. The macula is swollen. There are adhesions to the posterior surface of the cornea.

Microscopic: The iris and perforated lens are adherent to the scar of a perforating wound of the central cornea which includes pigment granules. There is anterior synechia and the anterior chamber contains serous exudate and pigment-bearing wandering cells as well as chronic inflammatory cells which also infiltrate the iris. There is massive infiltration of the ciliary body and choroid by lymphocytes with patches of epithelioid cells resulting in thickening of the ciliary body and the choroid at the disc. There are occasional giant cells but there is no destruction of tissue; epithelioid cells contain pigment granules. In at least one area of the choroid the pigment epithelium is disturbed with the formation of a Dalen-Fuchs' nodule. The retina is uninvolved except for lymphocytic periphlebitis and an occasional focus of epithelial cells. A few chronic inflammatory cells cling to the inner retinal surface in clumps and are present in a plastic cyclitic membrane. There is serous retinal detachment. The disc is slightly edematous and the lamina cribosa is depressed.

Diagnoses: Perforating wound, cornea and lens; traumatic cataract; retinal detachment; sympathetic uveitis.

Progress: Left eye remained active until November 20, 1946. Patient was discharged from the hospital November 27, 1946. Vision in left eye 20/120. She was discharged on Atropine 1% daily and was followed in the Eye Clinic at intervals from four weeks to three months. On her last examination October 27, 1947, vision OS 20/120, and left eye appeared quiet at that time.

DISCUSSION

Sympathetic ophthalmitis is a specific bilateral inflammation of the entire uveal tract of unknown etiology characterized clinically by an insidious onset, a progressive course with exacerbations and usually a disastrous termination; and pathologically by a nodular or diffuse infiltration of the uveal tract with lymphocytes and epitheloid cells which almost invariably follow a perforating wound involving uveal tissue.

The disease appears in the injured eye or exciting eye at a variable time after injury and affects the other eye at the same time or shortly afterwards, but the clinical and pathological picture presented by the

two eyes is identical. The general incidence of sympathetic ophthalmitis is difficult to assess since the cases from the single observers are few and pathological proof of the condition is usually lacking so that the diagnosis in many reported cases remain presumptive. It is, however, a relatively rare disease and owing to the improvements in the surgical technic of the treatment of wounds is undoubtedly becoming rarer.

Quite a number of conditions may be responsible for sympathetic ophthalmitis. In general terms, it may be said that of these the most common are perforating wounds which account for some 65 per cent of these case in literature; operative wounds are responsible for another 25 per cent, while the remaining 10 per cent are made up of cases which follow non-perforating contusions with sub-conjunctival scleral rupture, perforating corneal ulcers, and intra-ocular tumors. It is the rarest thing for sympathetic inflammation to arise without rupture of the globe.

It is a very old observation that suppuration in the injured eye is rarely followed by sympathetic disease, so old indeed that pre-Listerian surgeons intentionally produced the beneficent suppuration in a badly injured eye believing that the purulent inflammation destroyed the factors responsible for the condition or prevented infection passing up the optic nerve by sealing the lymph spaces.

The statistical incidence of sympathetic ophthalmitis after perforating wounds of the globe is of interest. Its assessment is somewhat difficult owing to lack of adequate proof of the condition in many instances. An average might be taken from literature at about 2 per cent.

The interval between injury and the onset of the inflammation is also of importance. This varies from nine days to twenty years. In general terms, however, it may be said that cases occur exceptionally before an interval of two weeks; that 65 per cent occur before an interval of two months; 80 per cent before an interval of three months and 90 per cent before the lapse of a year. The most dangerous time is from the fourth to the eighth week We may assume that the chances of development of sympathetic ophthalmitis after three months are few, but that the possibility of its occurring exists indefinitely.

In treating these conditions prophylaxis should be the first concern with adequate toilet of perforating wounds. The most common precursors of sympathetic disease are delayed healing owing to entanglement of the iris, ciliary body, or lens capsule in the wound and the retention of a foreign body. Every endeavor should therefore be made to eliminate these at the earliest possible moment as upon the success of these results, the ultimate fate may depend. If, despite these precautions the eye continues to remain irritable, with photophobia, lacrimation and ciliary injection, more drastic steps should be contemplated. It is now generally recognized and universally acted upon that the most valuable and important method to prevent the onset of sympathetic disease is the enucleation of the injured eye before the characteristic pathological changes have become established.

NEW ORLEANS
Medical and Surgical Journal

Established 1844

Published by the Louisiana State Medical Society under the jurisdiction of the following named Journal Committee:

M. D. Hargrove, M. D., Ex Officio
C. Grenes Cole, M. D., Chairman
E. L. Leckert, M. D., Vice-Chairman
Sam Hobson, M. D., Secretary
C. M. Horton, M. D.
Leon J. Menville, M. D.

EDITORIAL STAFF

Philip H. Jones, M. D.....*Editor*

COLLABORATORS—COUNCILORS

Edwin L. Zander, M. D.
Joseph S. Kopfler, M. D.
Guy R. Jones, M. D.
Paul D. Abramson, M. D.
George Wright, M. D.
W. E. Barker, Jr., M. D.
C. A. Martin, M. D.
O. B. Owens, M. D.

Paul T. Talbot, M. D.....*General Manager*

1430 Tulane Avenue

SUBSCRIPTION TERMS: $3.00 per year in advance, postage paid, for the United States; $3.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

THE JOURNAL *does not hold itself responsible for statements made by any contributor.*

POSSIBLE EFFECTS OF CHEMICALS USED IN PERMANENT WAVE PROCESS

It is estimated that some permanent wave process will be used fifty million times this year and that in the past three years thirty millon or more cold waves have been given. Recently the process and materials for "cold wave permanent" have been made available for use in the home, with the result that many persons will deal intimately with chemicals about which they know little.

The incidence of injurious effects from the "cold wave" process has been stated to be under 0.1 per cent. Goldman, Mason and McDaniel divide these effects into changes in the hair, skin irritations and body poisoning. The change in the hair include drying, splitting, changes in tensile strength elasticity; there may be darkening of the hair and it is stated that the hair is not in a normal state after the process.

In the "cold wave" process the hair is treated with an alkaline reducing agent, usually ammonium thioglycolate. Systemic poisoning from salts of thioglycolic acid has been studied experimentally. It was found that ammonium thioglycolate may be absorbed percutaneously and that minor fluctuations occur in the hemoglobin content of the blood and in the volume of erythrocytes of animals. However, the dose level is higher and the manner of contact different from which would prevail under conditions prescribed for use. Accordingly it was felt that the risk of systemic effects was remote if directions were followed.

The skin irritations are a source of concern and are more likely to be observed in the operator than in the patron. It is stated that none occurs in concentrations below 8 per cent by volume of ammonium thioglycolate. The effect on the skin appears when the solution is applied in undiluted form over a prolonged period of time. It is one of direct irritation and not sensitization.

The "cold wave process" involves the use of many substances. In the event of any irritation presumed to have followed the use of this process, the skin must be tested with the six or more solutions employed in the various steps. The skin lesions are described as pruritic, discrete papulopustular and vesicular about the scalp, ears and shoulder, or an eczematous contact dermatitis of the hair line and occasionally of the scalp.

A carefully devised procedure is recommended for purposes of prophylaxis. Among others, it is suggested that no large quantities of the cold waving materials be allowed to remain on the surface of the skin, and that as far as possible none should be permitted to remain longer than is re-

quired to remove it from such skin surfaces as those of the neck and forehead.

THE PARISH MEDICAL SOCIETY

The functioning unit of organized medicine is the county, or parish medical society. It is here that medicine first began to grow. The parish society is the place where the individual physician must be informed and kept united to the whole. By knowing his associates and their problems, a sense of solidarity is maintained. Where this exists a medical organization can speak with authority and effectiveness. At present more than at any time past such a forceful voice is needed.

However, many factors have contrived to weaken that voice. Prominent among these are specialization, specialty societies and hospital staff meetings. The physician's day does not stretch any more than that of his patients. As a result there may be more meetings than there are evenings to put them in. The hospital has a special call on the doctor's time; he is pushed by the rating authorities and pulled by an attractive program to attend hospital staff sessions. To secure a satisfactory attendance the program is frequently one of scientific essays and not hospital business. In this manner the hospital staff usurps the function of the parish society in many cities.

The meetings of hospital staffs and specialty groups have their function but the parish society represents and acts for all. It is here that medicine's public relations must be properly considered; it is here that plans for such things as publicity, medical emergencies, night calls, blood banks, epidemics and health programs should be discussed and understood. These matters are the concern of the membership and of the parish or county societies.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

MEETING OF HOUSE OF DELEGATES AMERICAN MEDICAL ASSOCIATION

Chicago, June 21-25, 1948

Dr. M. D. Hargrove, President and Members of Executive Committee, Louisiana State Medical Society, New Orleans.

Gentlemen:

The House of Delegates of the AMA was called to order on Monday, June 21, 1948, at 10:00 a. m. with both delegates from the Louisiana State Medical Society present. Dr. Graves was made a member of the reference committee on amendments to the Constitution and By-Laws. Out of 175 delegates 160 were present at the initial meeting.

The recipient of the distinguished service award was Dr. Isaac Arthur Abt, of Chicago.

The first session was taken up with the president's report and the introduction of the president-elect, followed by reports of the Board of Trustees and the Council. Dr. Ernest Howard, the new assistant secretary of the AMA, was presented to the group. Surgeon General Swanson, USN, gave a very interesting talk and Dr. Scott Stevenson brought greetings from the British Medical Society. This was followed by greetings from Dr. H. B. Washburn, president of the American Dental Association.

As usual there was much work to be accomplished in the House and our only off day (Wednesday) had to be filled with both morning and afternoon sessions. A tremendous amount of work was accomplished and of particular interest was the adoption of a new Constitution and By-Laws. This will be published shortly and we believe it

would be worthwhile for each member of our Society to receive a copy.

In addition a new "Principles of Medical Ethics" was presented. All in all we believe it is an excellent guide to correct conduct but there is one part to which we believe objection should be advanced and one morning was spent in a committee meetings giving reasons why this should be done. As soon as these are published not only every member of our Society but also every medical student graduating from our two universities in Louisiana should be given a copy.

A committee of five to study the nursing problem in the United States gave a very comprehensive report. It was interesting to see that from a questionnaire sent by the AHA in 1947 to 3800 member hospitals, of 2376 replies received, 37 per cent stated they needed no more nurses. This committee proposed training of nurses in the future under two grades: a. professsional nurses; b. trained practical nurses. The professional nurses to be divided into: a. nurse educators; b. clinical nurses. For trained practical nurses was recommended one year of training made up of three months theoretical and nine months practical training. This could be completed in a hospital or three months theoretical training taken under the Department of Education and nine months training in an acceptable hospital. Provisions are made to allow credits in select cases toward training for advancement from a group of trained practical nurses to the group of professional nurses.

The committee to study the Red Cross blood bank situation spent the entire morning in hearings and wound up with a very comprehensive report. It was the opinion of this committee that the "approved in principle" be construed as follows: 1. Local control must be by the county medical society. 2. The local medical society should be the initial contact contemplating inauguration of a new blood bank. 3. No publicity or news shall be released except by mutual consent of the local county medical society and the local chapter of the American Red Cross. 4. Difference of opinion in establishment or operation of a blood bank in either administrative or technical details shall be arbitrated at state level by joint committees of the state medical society and the American Red Cross. This committee also feels that any provision for free medical service or supplying to everyone, without regard to ability to pay, is in opposition to the principle that it is the responsibility of an individual to assume the obligation of medical expenses just as he does for other living expenses. It deplores the use of the term "free blood" in publicity of the American Red Cross.

A resolution was adopted opposing the plan of the American Academy of Pediatrics to seek federal support.

In the report of the Secretary the State of Louisiana was mentioned as having 42 component societies in 64 parishes of the state with 15 parishes not being organized. Of the 2601 doctors registered in Louisiana 1722 are members of the State Society and 876 are fellows of the AMA.

A motion was adopted that under the VA plans the veteran should be able to be treated in his own community and in a hospital and by a doctor of his own choice.

The Committee on Medical Education and Hospitals was increased from five to seven members with the provision that one of these members should be a practicing physician, not a member of a teaching staff of a medical school and not associated with a medical school hospital staff.

The AMA and the American Cancer Society were requested to adopt standards for cancer detection and diagnostic clinics.

A resolution was also adopted urging the state medical societies to make wives of their members automatically members of the auxiliary and that these state societies find some way to finance the dues of the national auxiliary organization.

The following officers were elected after Dr. Sensenich was installed as president:

President-elect—Dr. Ernest E. Irons, Chicago

Vice-President—Dr. R. W. Fouts, Omaha

Secretary—Dr. George F. Lull, Chicago

Treasurer—Dr. J. J. Moore, Chicago

Speaker of House of Delegates—Dr. F. F. Borzell, Philadelphia

Vice-Speaker of House of Delegates—Dr. James Reuling, Bayside, New York.

The new members of the Board of Trustees elected were: Dr. Ed. Hamilton, of Illinois, to succeed Dr. Irons; Dr. Gunnon Gunderson, of Wisconsin, to succeed Dr. Braasch; Dr. Walter B. Martin, of Virginia, to fill the unexpired term of Dr. C. W Roberts, deceased.

It is the suggestion of your delegates that when the complete reports, particularly those of the Committee on National Emergency Medical Service, and the Committee on Rural Medical Service, appear in the Journal of the AMA that it will be time well spent to read these in their entirety.

Respectfully submitted,

VAL H. FUCHS, M. D.
J. Q. GRAVES, M. D.
Delegates to AMA.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

ALVARENGA PRIZE

In recognition of his studies on "Sludged Blood" the College of Physicians of Philadelphia awarded on July 14, 1948, the Alvarenga Prize for this year to Melvin H. Knisely, M. D., of the University of Chicago.

The Alvarenga Prize was established by the will of Pedro Francisco da Costa Alvarenga of Lisbon, Portugal, an Associate Fellow of the College of Physicians of Philadelphia, "to be awarded annually by the College of Physicians on each anniversary of the death of the testator, July 14, 1883."

The College usually makes this award for outstanding work and invites the recipient to deliver an Alvarenga Lecture before the College.

AMERICAN COLLEGE OF PHYSICIANS

The American College of Physicians will conduct its 30th Annual Session at New York, N. Y., March 8 through April 1, 1949. Dr. Franklin M. Hanger, Jr., of New York City is the Chairman for local arrangements and the program of clinics and panel discussions. The president of the College, Dr. Walter W. Palmer, Director of The Public Health Research Institute of the City of New York, Inc., and Professor Emeritus, Columbia University College of Physicians and Surgeons, is in charge of the program of morning lectures and afternoon general sessions.

RESEARCH FELLOWSHIPS

The American College of Physicians announces that a limited number of Fellowships in Medicine will be available from July 1, 1949-June 30, 1950. These fellowships are designed to provide an opportunity for research training either in the basic medical sciences or in the application of these sciences to clinical investigation. They are for the benefit of physicians who are in the early stages of their preparation for a teaching and investigative career in Internal Medicine. Assurance must be provided that the applicant will be acceptable in the laboratory or clinic of his choice and that he will be provided with the facilities necessary for the proper pursuit of his work. The stipend will be from $2,200 to $3,200.

Application forms will be supplied on request to The American College of Physicians, 4200 Pine Street, Philadelphia 4, Pa., and must be submitted in duplicate not later than November 1, 1948. Announcement of the awards will be made as promptly as is possible.

PHYSICIAN WANTED

Due to the recent death of Dr. R. K. Comeaux, of Youngsville, there is need for a physician in that community. Due to the volume of practice a young, active physician would be most suitable and a knowledge of the French language would be quite

advantageous. Office space and equipment is available. For further information contact Mrs. R. K. Comeaux, Youngsville.

DR. GILBERT CHRISTIAN ANDERSON

The medical profession lost one of its most widely esteemed and useful members in the death of Dr. Gilbert Christian Anderson, past president of the Louisiana State Medical Society and a pioneer in the practice of neurosurgery, who passed away on June 27, 1948. Dr. Anderson was a graduate of Columbia University, later seeing service in World War I.

Upon the completion of his specialized training in neurosurgery at the Mayo Clinic he came to New Orleans in 1927 as the first practicing neurosurgeon in this area. The abundant enthusiasm and tireless energy which he carried to his classes as professor of neurosurgery at Tulane University Medical School, and later at L. S. U. Medical School, inspired and encouraged others to enter the field of neurosurgery.

Dr. Anderson was above all else a devoted physician, and he was every ready to help the poor as well as the more fortunate. He gave his time to the out-patient clinic at Touro Infirmary for 18 years, and later he was available when his special talents and knowledge were needed.

At De Paul Sanitarium, where he was consultant in neurosurgery for a number of years, there were many problems resolved by his unusual diagnostic talents.

WHEREAS the entire Staff and personnel of the De Paul Sanitarium are conscious of a tremendous loss in the passing of this greatly admired member of our group:

AND WHEREAS we wish to join with the Sisters of Charity of De Paul Sanitarium in praying that God will reward Dr. Anderson with eternal peace and happiness for his great and noble service to his fellowmen,

BE IT RESOLVED in recognition and appreciation of his able, unselfish and devoted service to the mentally ill, the entire Staff and personnel of De Paul Sanitarium express their consciousness of a tremendous loss in the passing of this greatly admired member of our group.

BE IT FURTHER RESOLVED that a copy of these resolutions be sent to the family of Dr. Anderson, a copy be published in the New Orleans Medical and Surgical Journal, and a copy spread on the minutes of the De Paul Staff.

HENRY O. COLOMB, M. D.
EDMUND CONNELY, M. D.
CHARLES S. HOLBROOK, M. D.

WOMAN'S AUXILIARY

A new Council has been formed in the State Auxiliary with Mrs. D. B. Barber as Chairman and a representative from each district. It is to be known as the *Auxiliary Council on Medical Service and Public Relations.* The purpose is to serve in conjunction with the Louisiana State Medical Society's Council on Medical Service and Public Relations and to act as a news gathering agency for our own publication, *"Auxiliary News and Views"*, appropriately named, sponsored and mailed by the Society Council.

It was this new Council's privilege to meet in New Orleans July 10, 1948 with the Louisiana State Medical Society's Council on Medical Service and Public Relations. Dr. A. V. Friedrichs, Chairman, presided and outlined their program so far and the activities planned for the ensuing year.

Dr. Friedrichs says in the booklet on Public Relations Program, "The purpose of this program shall be to further a better understanding of problems both to the laity and the profession, to disseminate information which is of general intent and value through all available media, and to keep both the public and the medical profession informed of significant progress in all fields of medicine." This booklet and one entitled, "Services of Your Medical Society" have been sent to all Louisiana doctors as well as to other state societies. A one page *News letter* is mailed monthly to every doctor. Ask your husband to bring them home and share them with you!

The Society Council has attended and actively participated in many meetings including, The Louisiana Rural Health Council, the Steering Committee of the Health, Physical and Safety Education Council of the Department of Education, Health Workshops at Southwestern Louisiana Institute, Lafayette and Northwestern State College, Natchitoches, local, state and national medical meetings and also the National Health Assembly in Washington, D. C.

Splendid cooperation has been given by nine radio stations throughout the state with a total of 168 broadcasts already presented at no cost to the doctors.

Society and Auxiliary members are urged to continue in getting information of these broadcasts before the public by placing posters in your office, drug stores and other places. Doctors are requested to enclose information supplied them in their monthly bills. As part of the new program newspapers are prepared to publish weekly a health column on timely articles supplied them by this Council.

A *Speakers Bureau* has been set up and each Society should select five doctors in each community and send their names into the state Bureau along with a list of clubs and organizations. These lay organizations ordinarily using speakers will be notified that talks on medical subjects and speakers will be available through this Bureau. Speakers' kits will be supplied the doctors. A *News Bureau* has been set up on all matters of public interest and Auxiliary members are requested to send in

any information on problems or plans of a local interest to Mr. Frank Lais, Executive Secretary, 1430 Tulane Ave., New Orleans. Mr. Lais reported on the progress of the Voluntary Prepayment Surgical Care Plan.

Most significant of all the public relation endeavors is the new School Program. The Council on Medical Services and Public Relations proposes to assist in the establishment of HEALTH COUNCILS throughout the State of Louisiana. It has already participated in the organization of a STATE HEALTH COUNCIL. Plans are now underway in its participating in the establishment of parish health councils and possibly the establishment on individual community health councils. A doctor should be at the head of each Health Council and it is their aim to see that every school child receives a physical examination before he or she enters school. This State Health Council is working with the Farm Bureau and the State Department of Education and others. Much work is yet to be done. Let us all do our share, starting now. Get in touch with your State President, Mrs. O. B. Owens for further information.

MRS. F. U. DARBY,
Press & Publicity Chairman.

BOOK REVIEWS

Coronary Heart Disease: By A. Carlton Ernestene, M. D. Springfield, Charles C. Thomas, 1948. Pp. 95. Price, $2.50.

This small book of eighty-six pages is divided into eight chapters with five pages of references.

In presenting the different manifestations of coronary disease the views expressed may be said to represent the present status of our knowledge of this disease.

In the introductory chapter the author calls attention to the fact that in many cases of coronary disease the diagnosis will depend entirely on the patient's history. This is not new but will bear repeating. The fundamental pathological changes usually found in coronary disease, the influence of hypertension and diabetes is discussed briefly but sufficient for a book of this size.

In discussing the differential diagnosis of angina pectoris the author states that, "The pain in hiatus hernia, esophogospasm or cardiospasm is not brought on by physical exertion but may be induced by excitement or large meals and is usually of longer duration than is angina pectoris and it is not relieved by rest but nitroglycerine may be effective." The opinion of the reviewer is that this is true as a rule and should be of help in the differential diagnosis. There are, however, cases of angina pectoris where the attacks seem to be precipitated more by emotional stress than by physical exertion.

It is stated that in myocardial infarction that the electrocardiogram will show diagnostic changes in practically every case if the records are made every few days and precardial leads are used in addition to the standard leads. It should be remembered that a sufficient number of chest leads should be made. Most of the important findings in the differential diagnosis of myocardial infarction are mentioned, but some of importance are not. To mention one; the diastolic murmur heard in the aortic area in cases of dissecting aneurysm of the aorta.

Three pages are devoted to "acute coronary failure". This may be a convenient name for cases of cardiac pain that last twenty minutes or longer not followed by evidence of infarction. However, the line of demarcation between these cases, angina pectoris and myocardial infarction is not clear clinically.

In speaking of the recent emphasis given to certain harmful effects that may follow the sudden enforcement of recumbency in patients who have congestive failure, the author has this to say, "These effects are to be regarded as complications against which suitable precautions should be taken and the possibility of their occurrence does not detract from the importance of rest in heart failure." This is true and to the point. The reviewer feels that possibly the recent emphasis given to the complications that arise in bed patients has been misunderstood by some.

Some of the newer remedies such as plasma and dicumarol are discussed.

There may be slight differences of opinion in some of the views expressed in this book but there are few. The book is well written, the print is good. The book should be of value to anyone interested in the subject. The reviewer recommends it.

J. M. BAMBER, M. D.

The Acute Bacterial Diseases, Their Diagnosis and Treatment: By Harry Filmore Dowling, M. D., F. A. C. P. Philadephia, Saunders, 1948. Pp. 465. Price, $6.50.

This volume attempts to summarize modern concepts and methods used in the diagnosis as well as treatment of coccal and bacillary infections or the intoxications due to their respective exotoxins. Early sections review the basic features of the clinical characteristics and laboratory diagnosis of infectious diseases. These are followed by a chapter dealing with general measures employed in treatment such as administration of oxygen and fluids, control of food and diet, support of circulation, isolation procedures. Separate chapters next

discuss the use of antisera, sulfonamides, penicillin and streptomycin respectively, with stress on their administration, absorption, distribution, excretion and toxicity. Thereafter the clinical conditions caused by the various etiologic agents are presented. For each disease there is a concise statement of the pathogenesis, general signs and symptoms, special features of the infection and likely complications, methods for differential clinical diagnosis, laboratory examinations, methods for treatment and prevention.

There are numerous illustrative charts and summaries of typical case histories, tables of pertinent statistical data, well-reproduced photographs and drawings of characteristic lesions. An appendix outlines methods for the assay of sulfonamides, penicillin and streptomycin in body fluids. The authors and publishers deserve credit for their success in combining literary and graphic methods to present effectively a large amount of useful information.

The book is intended as a practical guide for physicians. In view of Dr. Dowling's stress on the importance of etiologic diagnosis, one criticism which may be fairly levelled is the sketchiness of the presentation concerning laboratory examinations throughout most of the book. The information on the collection and handling of specimens is often perfunctory and is rarely adequate to enable the physician to evaluate the performance of the laboratory doing his work or to suggest the most appropriate methods for a particular examination.

In the case of certain infections, however, the reader might obtain an erroneous notion of present-day concepts of the immunology or proper bacteriological practice. Since the book should have a wide sale it is to be hoped that the next edition will be revised to remedy the presently existing defects.

MORRIS E. SHAFFER, M. D.

Laboratory Diagnosis of Protozoan Diseases: By Charles Franklin Craig, M. D., M. A. (Hon.), D. Sc. (Hon.), F. A. C. S., F. A. C. P., Colonel, M. C., U. S. A. (Retired), 2d ed. Phildelphia, Lea and Febiger, 1948. Pp. 384. Price, $6.50.

This is a thoroughly revised and enlarged sequel to the praise-worthy first edition which was published in 1942. There is evidence that the War years contributed not only considerable new information but added materially to practical methods for the laboratory diagnosis of the protozoan infections.

The volume is divided into six parts as follows: I, Amebiasis and Intestinal Flagellate Infections; II, The Leishmaniases (Kala-Azar, Oriental Sore and Espundia); III, The Trypanosomiases (African Trypanosomiasis and Chagas' Disease); IV, Coccidiosis; V, Malaria, and VI, Balantidiasis. In each infection the etiological agent is clearly described and illustrated, the methods for obtaining diagnostic material are listed and the technics for carrying out useful diagnostic procedures are presented in detail. For the physician or medical technologist with a basic knowledge of the protozoa of man and of laboratory methods of diagnosis Colonel Craig's handbook constitutes a most valuable, authoritative aid.

The book has an attractive format, the print is clean and the illustrations, including line drawings, half tones and seven color plates, are pleasing in appearance and diagnostically valuable. The binding is neat and substantial. Both author and publisher are to be complimented on the new edition. The reviewer recommends this volume to all persons who have professional interest in protozoan diseases of man.

ERNEST CARROLL FAUST, PH. D.

Headache and Other Head Pain: By Harold G. Wolff. New York, Oxford University Press, 1948.

In this scholarly work Wolff discusses the various types of head pains. There is first a preliminary description of pain, in which the various factors; causes, threshold, references, etc. are ably discussed. Wolff incidentally was President of the Association for Research in Nervous and Mental Disease. With his coworkers in this association he has added greatly to our knowledge of the unpleasant sensations which we call pain. The group published a collection of reports in 1943 in a volume entitled "Pain".

He is a neuropsychiatrist, who has for many years been interested in various types of headaches, and much of our knowledge of the various headache syndromes has been derived from his work. There are many articles on this subject which he has contributed, either alone or with others. He has made intricate studies on the localization of various head pains, along with experimental studies on the associated pathological physiology involved. This book is of great value principally because this work is now organized and unified in one publication, and the assembled data are of great help to anyone interested in the problem of headache.

The views of the author however are basically those of a neurologist and psychiatrist. The reviewer does not accept for instance the stand that Dr. Wolff has taken on the uestion of allergic headaches. The author feels that psychogenic factors are of paramount importance in certain vascular pains, such as the entity spoken of as migraine. We all agree that migraine is but one variant in the larger group of vascular headaches. According to him the causes of these pains are due to mental states, rather than to somatic alterations.

He states that "no well controlled clinical study has yet been reported to support the contention

that migraine headache is an allergic reaction." Yet he goes on to show histamine has a very definite vasodilating effect on intra and extracranial vessels. We now realize that histamine production in the body is part of the allergic reaction.

In support of his view he cites Pfeiffer, Dreisbach, and Roby. The following is a verbatim quote from the footnote on pages 360-61.

"To illuminate further this matter of food 'allergy', the following experiment was performed. Four able physicians, experienced in experimental methods, and themselves migraine patients, were the subjects. These four physicians, who were of the opinion that they could predictably produce migraine headache in themselves by eating chocolate in any form and in minimal amounts, were each given a set of lettered, sealed envelopes, with the key to the contents held in the laboratory, and unknown to the subject. Each envelope contained either 8 Gm. powdered chocolate or 8 Gm. lactose, in 8 black capsules. The two sets of capsules were indistinguishable in appearance. Two subjects ingested the contents of an envelope at convenient intervals. One subject ingested the contents of an envelope at regular intervals, three times a week. The fourth subject who commonly awoke on Saturday mornings with a migraine headache attack after eating chocolate on Friday evening, ingested his capsules on Friday evenings for a period of four months. All were instructed to include no chocolate in their regular diet. Careful records were kept by each subject, including the following data: the letter on the envelope; the date and time of ingestion of the contents; the date and time of onset of all headaches experienced during the experimental period.

"It was found in these subjects that headaches sometimes followed the chocolate, sometimes the lactose, but most commonly attacks occurred without reference to the ingestion of capsules. Migraine headaches followed the ingestion of lactose just as frequently as they followed the ingestion of chocolate. The data thus accumulated indicate that in these individuals who considered themselves 'allergic' to chocolate even in minimal amounts, the occurrence of their headaches was no more related to the ingestion of chocolate than it was to the ingestion of lactose."

To the reviewer this does not prove that an allergic reaction is not capable of producing the various vascular dilatations which cause certain headaches. It is not conceivable that the wealth of evidence to support the allergic concept is erroneous, and that the headaches due to certain antigens are all explainable on another basis. Too many patients have stated that certain foodstuffs regularly produce headaches. The widespread skepticism concerning the phenomena of clinical allergy is well illustrated here.

The allergist recognizes clearly that psychogenic factors alone may precipitate these vascular changes. The headache patient has a substrate or tendency to vascular dilatation of the cranial vessels. This dilatation may be brought on by vasodilators such as the nitrites. In the allergic individual we see the endogenous production of a potent vasodilating substance. Also the allergic individual is as a rule unstable, in that he is affected by non-specific factors which may initiate the syndrome. Upsets in the bodily physiology, environment, mental state, etc. are often capable of setting the vasodilating mechanism into action.

Also at times the allergic individual will not be affected by the allergenic substance, if the optimum situation is not present. We know that true allergic asthmatics may tolerate the causative antigens under certain conditions. In some patients, there is also the quantitative factor to be considered. As an example a small amount of a certain foodstuff may be tolerated. A larger amount may precipitate symptoms.

The quantity of histamine required to produce symptoms is an important factor. We all recognize that too small an amount may not affect the vessels.

This is also true of other vasodilators. A physician subject to migraine, knows by experience that he can tolerate a small amount of an alcoholic beverage. Several drinks on the other hand, quickly produce an annoying headache.

In support of Dr. Wolff's viewpoint it must be stated that he and others have found that various therapeutic measures give varying degrees of relief to individuals with migraine. Their measures may be of a more or less non-specific nature. He discusses dietary methods in the management of migraine under the following heading: "Agents or Procedures That Act by Inducing Feelings of Security through Suggestion, Reassurance, and Confidence in the Well-Planned Regimen Imposed by the Sanitarium or Physician". It is obvious that these patients are helped by almost any vigorous, directed type of therapy. This however still does not prove that the allergic reaction is not capable of initiating an attack.

Dr. Wolff concedes that headache may result if the nasal mucosa is involved in the allergic reaction. He however does not emphasize the frequent occurrence of frontal pain which is often seen in conjunction with nasal allergy. In my experience this very frequently complicates hay fever or perennial rhinitis.

Psychiatrists have a tendency to explain the various allergic syndromes on a basis of conflicts. However, the formation of specific antibodies is a somatic process. I cannot accept the idea that a house dust asthmatic, who has transferable humoral antibodies to house dust, can be classified in any way except as a house dust allergic. Under control conditions, asthma may be precipitated by giving the patient a large amount of house dust

extract. It is granted that emotional upsets may initiate individual attacks, but the underlying sensitization or substrate is in the background. Mental conflicts may be demonstrated in patients with cancer, appendicitis, asthma, etc., but this does not prove the mental disturbance is the essential cause of the disorders. I have been informed by a sincere psychiatrist that the formation of specific antibodies themselves may have a psychogenic basis. Does anyone claim that the antibody formation associated with immunity is in any way connected with psychogenic factors? It has been stated that allergy is really an altered form of immunity. Therefore, this theory is not logical and cannot be accepted.

Exclusive of this difference of opinion, I found Dr. Wolff's book to be of great interest, and as stated above feel that it should be read by any physician interested in the problem of headache.

HENRY D. OGDEN, M. D.

Diseases Transmitted from Animals to Man: By Thomas G. Hull, Ph. D., 3rd ed. Springfield, Charles C. Thomas, 1947. Pp. 571. Price, $10.50.

Commenting on the second edition of this work in 1941, the late Dr. Musser, wrote—"The book is filled with information which would be of service to the doctor of medicine, of veterinary medicine, and to public health officials, as well as, to lay persons who are interested in individual and public health problems. For the physician there is a tremendous amount of information condensed in a relatively few pages which would necessitate an extensive survey of the literature were he to attempt to obtain facts such as are contained in this excellent book. It is to be recommended most highly". The present edition lives up to this recommendation. All the chapters of the old edition have been rewritten and brought up to date and nine new chapters added. Of these latter, the one on yellow fever is the most complete and informative. Tuberculosis has been expanded from 34 pages to 56 and is very complete. Section 5, includes at the end, a list of the diseases transmitted to man by the horse, cow, dog, cat, etc. These lists are indeed formidable for each genus. There are 30 new illustrations.

While the work is primarily of interest to public health people, the physician will undoubtedly find it a mine of information, but with scant references to treatment. Except for one reference to *Amoeba histolytica* noted misprints are remarkably few. Such a book should be available to all those who have the overall supervision of the health of sections of the population in any part of the world.

A. J. WALKER, M. D.

The Pathology of Nutritional Disease: By Richard H. Follis, Jr. Springfield, Charles C. Thomas, 1948. Pp. 291. Price, $6.75.

This book fills a real need in bringing together, under one cover, existing information on the pathology of nutritional disease in Mammalia. The author discusses both physiological and morphological changes which result from deficiency of each of the known essential nutrients including elements, amino acids, vitamins and fatty acids. A description of the pathology which occurs in man is included whenever facts are available. The paucity of information particularly in humans is astounding and should stimulate further research. No single nutrient has been studied simultaneously from a biochemical, morphological and physiological standpoint.

The book includes a number of excellent photographs illustrating morphological changes and a valuable bibliography of 791 references. It should be of great assistance to students of nutrition and related sciences.

GRACE A. GOLDSMITH, M. D.

PUBLICATIONS RECEIVED

Blakiston Company, Philadelphia: Diagnosis in Gynaecology, by James V. Ricci, A. B., M. D.

Grune & Stratton, New York: Failures in Psychiatric Treatment, edited by Paul H. Hock, M. D.; Progress in Neurology and Psychiatry (Volume III), edited by E. A. Spiegel, M. D.; Venous Thrombosis and Pulmonary Embolism, by Harold Neuhof, M. D.

C. V. Mosby Company, St. Louis: Clinical Laboratory Methods and Diagnosis (Volumes I, II, and III, 4th edition), by R. B. H. Gradwohl, M. D., D. Sc., F. R. S. T. M. & H. (London); Handbook of Ophthalmology, by Everett L. Goar, A. B. M. D., F. A. C. S.; Practice of Allergy, (2nd edition), by Warren T. Vaughan, M. D., revised by J. Harvey Black, M. D.; Principles Governing Eye Operating Room Procedures, by Emma I. Clevenger, R. N.

Philosophical Library, New York: Twentieth Century Speech and Voice Correction, edited by Emil Froeschels, M. D.

W. B. Saunders Company, Philadelphia: Modern Clinical Psychiatry (3rd edition), by Arthur P. Noyes, M. D.

Charles C. Thomas, Springfield, Illinois: Intracranial Tumors (2nd edition), by Percival Bailey.

Williams & Wilkins Company, Baltimore: Gynaecological and Obstetrical Anatomy (2nd edition), by C. V. F. Smout, M. D., M. R. C. S. and F. Jacoby, M. D., Ph. D.; Medullary Nailing of Küntscher, by Lorenz Böhler, M. D., translated from the Eleventh German Edition by Hans Tretter, M. D.